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WITKOP: A SYNONYM FOR FAVUS

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'Witkop' (white head) is the descriptive term used in South Africa for a peculiar condition of the scalp occurring in the Bantu. The term was first introduced by McArthur and Thornton[1] in 1911 when describing syphilis amongst the Bechuana in the northern Cape Province. The relationship of witkop to syphilis is still a matter for discussion although several papers have appeared on the subject over the last 40 years. The condition has been regarded by some authors as a pustular syphilide, while others have regarded it as a favus infection. McArthur and Thornton,[1] who first described the condition, are emphatic that it is a manifestation of syphilis and they reiterate this opinion in a later publication.[2] Marshall and Wilson[3] state that it is found only in syphilitic South African Natives. Mitchell and Robertson,[4] in the second publication on witkop in the South African literature, definitely state that it is a favus infection. All writers on the subject are agreed that, while the condition occasionally occurs in other parts of Southern Africa, its greatest incidence is in Bechuanaland. It is also stated to be commoner among males than females and to occur most commonly during childhood up to the age of puberty.

In 1953, during a survey of 35,043 Bantu in the Bakwena Reserve of the Bechuanaland Protectorate,[5] we had the unique opportunity of observing the exact incidence of this condition and of examining the serological status of the patients. In all, we encountered 815 cases of witkop, on 348 of whom serological tests were carried out. As a result of our investigations we are firmly convinced that this condition is a favus infection of the scalp and is not a syphiloderm.

CLINICAL FEATURES

Witkop affects the scalp and usually commences between the age of 2 and 10 years. It rarely commences after puberty. Cases are occasionally seen in adults but, as a rule, in such

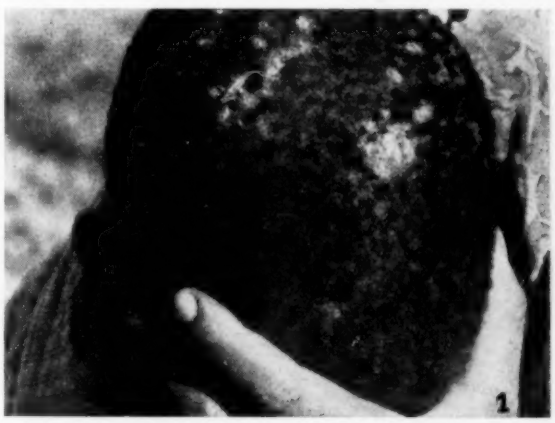

Fig. 1. Early stage of witkop, showing isolated greyish crusted exudate.

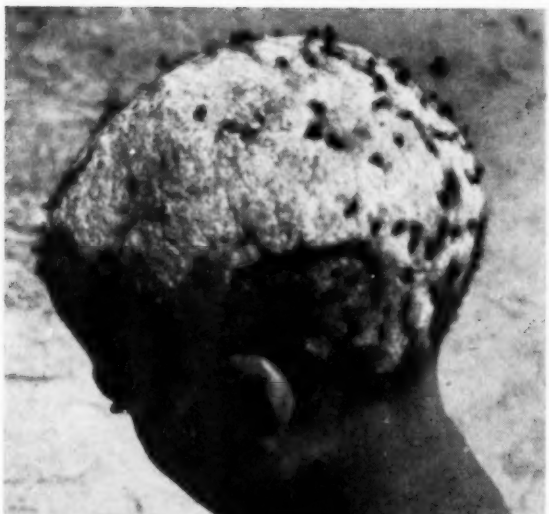

Fig. 2. More advanced case of witkop, showing confluence of exudate.

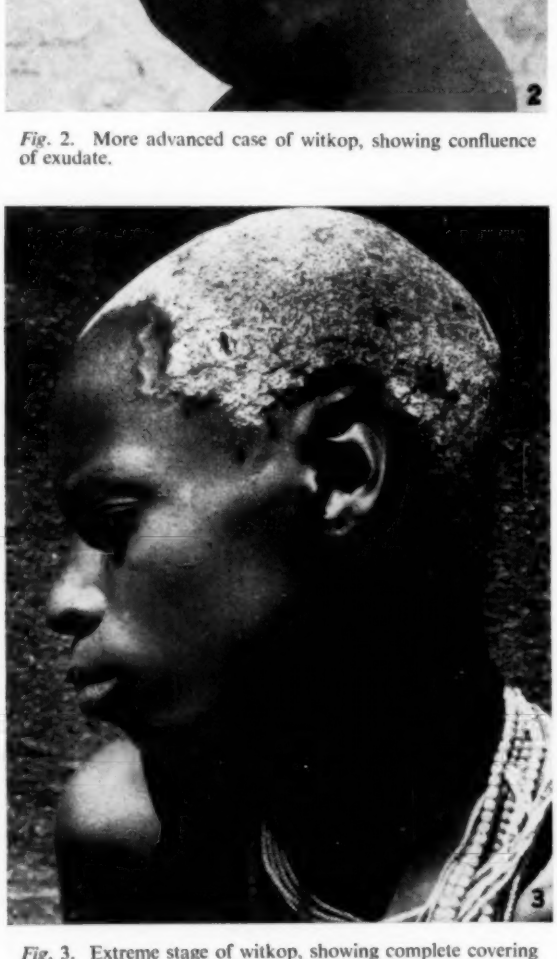

Fig. 3. Extreme stage of witkop, showing complete covering of scalp by greyish-white crusts and the presence of secondarily infected lymph glands.

cases the disease has commenced in childhood and has continued into adult life. The first sign is the appearance of small white, discrete, slightly raised crusts overlying indurated papules on the scalp (Fig. 1). The crusts spread and coalesce until eventually the whole scalp becomes a continuous mass of greyish-white crusted material. As the disease progresses the hair falls out leaving only isolated tufts (Fig. 2). Eventually only a fringe of hair remains in front, at the sides, and at the back of the scalp (Fig. 3). Removal of the crusts reveals a raw red surface which quickly becomes covered with exudate again if treatment ceases. The condition may persist for many years but there is a natural tendency to recover as the patient reaches adult life. In such cases areas of alopecia remain and, if the disease has become extensive, the patient becomes entirely bald, a condition to which the term 'kaalkop' has been applied. Secondary infection is usual and the occipital lymph glands become enlarged (Fig. 3).

ETIOLOGY

The assumption that witkop is a manifestation of syphilis appears from the literature to be based upon 5 observations:

(*a*) It is frequently associated with positive serological tests for syphilis.

(*b*) It is often found in patients who show clinical evidence of syphilis.

(*c*) Antisyphilitic treatment may lead to a remission of the scalp condition.

(*d*) Some authors have been unsuccessful in isolating the fungus.

(*e*) There is an apparent low infectivity of the condition.

During our observations in Bechuanaland we were enabled, because of the large number of cases with which we were dealing, to test the validity of these points. From our observations we reached the following conclusions in relation to them:

(*a* and *b*) Positivity was present in 40·2% of 348 cases of witkop in which serological tests for treponematosis were carried out. This compared with 37·0% seropositivity in the general population. The difference is not statistically significant. A high seropositivity rate is normally found amongst the Bantu of Southern Africa. In any random series of witkop or any other condition in the Bantu, therefore, a relatively high seropositivity rate as compared with Europeans will be found. This has been interpreted as evidence of the syphilitic nature of the witkop whereas, in fact, the findings are purely coincidental.

Schechter[6] makes a bold statement with which we are certainly not in agreement, viz. that witkop is always accompanied by syphilitic stigmata and responds to arsenical treatment. In surveying the literature on endemic syphilis from other parts of the world we have not found that witkop has been reported as one of its manifestations. Guthe,[7] however, states that Grin observed a few cases of witkop in Bosnia during his survey of endemic syphilis, but again this is explicable on the basis of the association of two diseases which tend to occur under conditions of poor hygiene. It is not to be disputed that witkop is frequently found in cases of active or latent syphilis, but in our experience in the Bakwena Reserve scabies is even more frequently found in association with endemic syphilis; this does not suggest that scabies is syphilitic in origin. Witkop and endemic syphilis are diseases which occur in conditions of poverty and poor hygiene and are, therefore, frequently found in the same patient, but this is not to be interpreted as evidence that witkop is a manifestation of syphilis.

(*c*) Fraser[8] states that witkop responds to antisyphilitic treatment but the follow-up in his cases was very short. Mitchell and Robertson[4] state that a full course of antisyphilitic treatment has no effect on the scalp condition. Marshall and Wilson,[3] in their 3 cases, observed improvement with arsenical and bismuth treatment

but they also applied local treatment. Their longest follow-up appears to have been 2 months. Schechter[6] dogmatically states that the condition yields to arsenical treatment but he gives no particulars as to whether local treatment was also used, or the length of time during which the cases were observed after treatment. McArthur[2] found that his early cases responded to antisyphilitic treatment but that the chronic cases were very obstinate. Our own experience has been that all chronic cases proved completely resistant to antisyphilitic treatment apart from the response of the secondarily infected glands. All, however, responded to local treatment with ointments with a degree of success which was directly related to the intensity and duration of the treatment. If treatment is thorough the condition responds, but with cessation of treatment it returns and in a short time the thick greyish white crust of exudate reappears. Early cases show some response to P.A.M. but we attribute this to control of the secondary infection and the tendency of the condition to heal spontaneously when conditions of general health and hygiene are improved.

(d) Trichophyton (*Achorion*) *schoenleini* has been successfully isolated by us from the scalp in the majority of the cases of witkop in which this has been attempted.

Sections of the scalp (Fig. 4) show the presence of a seropurulent and serosanguinous exudate on the surface. The epidermis shows hyperkeratosis, a few small foci of parakeratosis, acanthosis, spongiosis and intracellular oedema of the more superficial prickle cells. The papillae are oedematous and are infiltrated by acute inflammatory cells which extend into the overlying epidermis. In the upper dermis there is a diffuse subacute or chronic inflammatory reaction. In the mid-lower dermis there is a slight perivascular and perifollicular infiltration by chronic inflammatory cells. On the surface of the skin there is a dense mass of tangled, branching, septate hyphae. In the more superficial layer of this mass round or oval spores can be seen lying singly or in clumps. The fungal hyphae extend downwards into the hair follicles almost to the hair roots. In the upper half of the follicle they can be seen both inside and outside the hair shafts but in the lower half they appear to be entirely within the hair shafts (Fig. 5).

(*e*) A low degree of infectivity is a feature which witkop shares with favus[9] and it does not seem that this should be regarded as evidence against the condition being fungal in origin. Our own experience confirms the low infectivity of the condition, although more than one case has been observed in a family and McArthur[2] refers to 12 cases in 13 persons in two generations of one family. He interprets this, however, as evidence for the syphilitic origin of the disease and not for a fungal origin, for the members were widely scattered.

Our survey has enabled us to observe a larger number of witkop cases than has previously been reviewed and to observe the disease in relation to the occurrence of endemic syphilis. Serological tests (VDRL and Kolmer Cardiolipin Wassermann) were positive in 40·2% of 348 cases of witkop in which they were carried out. Of the total of 815 cases seen, 437 occurred in females and 378 in males, a ratio of 1.2:1 as compared with a ratio of 1.4:1 in the population as a whole. Over 90% of the cases occurred between the ages of 1 and 15 years. Our series shows no male preponderance of cases and no causal relationship between syphilis and witkop has been demonstrated. It is our opinion that witkop is not a manifestation of syphilis but that it is a favus infection of the scalp. The reason that it is so commonly associated with the seropositive state is purely epidemiological; it is, like endemic syphilis and scabies, a disease of the less hygienic members of the community.

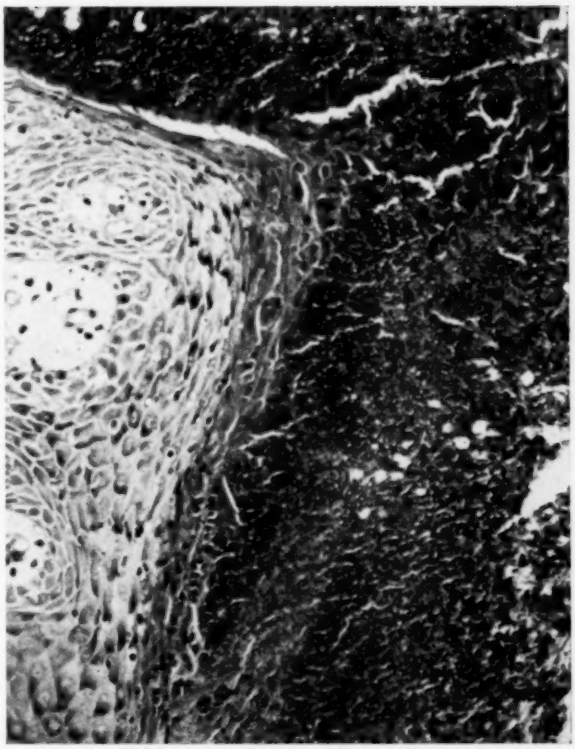

Fig. 4. Thick mass of seropurulent and serosanguinous exudate in which abundant branching septate hyphae are embedded. × 200 H & E.

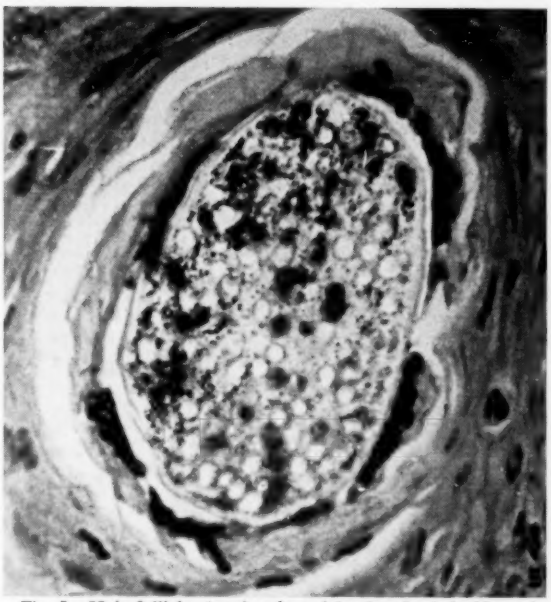

Fig. 5. Hair follicle showing fungal hyphae both inside and outside the hair shaft. × 750 periodic acid Schiff.

DISCUSSION

In describing endemic favus in Kentucky, Barrett[10] states that his series of cases conform with the following salient facts as stated by previous writers:

(*a*) The disease rarely appears after the 17th year,

(*b*) Favus is a disease of the country rather than the city.

(*c*) Frequently some children in a family fail to contract the disease even when conditions seem most favourable for doing so.

(*d*) Favus is a disease of the poor and undernourished.

(*e*) There is a tendency in adults towards latency, or spontaneous cure.

Witkop in Bechuanaland manifests the same features as Barrett's series of favus cases and it would appear that, not only can *T. schoenleini* be isolated from both conditions, but they parallel one another in their epidemiological and clinical features. Anderson[11] states that favus has been gradually diminishing as a problem for many years in European and American dermatology and that better living conditions and improved hygiene will soon bring an end to endemic favus in America. Our experience with witkop in Bechuanaland shows that it occurs most commonly amongst the poorest sections of the community. Those who live in grossly unhygienic conditions on the border of the Kalahari desert most commonly exhibit the condition. It gradually decreases in incidence in the more developed parts of the country, where improved water supplies, better economic conditions and the introduction of schools and medical facilities have improved the general hygiene of the community. There seems no doubt that, with improving hygiene, the condition will gradually disappear from Bechuanaland in the same way that endemic syphilis and scabies tend to disappear as living standards improve.

Witkop has apparently always been commoner in Bechuanaland than in other parts of South Africa. This may be due to the semi-arid conditions which prevail over the greater part of the country, as a result of which water is at a premium and personal hygiene at a discount. The tendency for witkop steadily to increase in incidence from the eastern part of the Bechuanaland Protectorate, with its moderate water supplies and rising standards of hygiene, to the western part, where semi-desert conditions prevail, supports this contention.

SUMMARY

815 cases of witkop have been observed during a survey of 35,043 Bantu in the Bechuanaland Protectorate. As a result of the investigations it is concluded that the condition is due to infection with *T. schoenleini* and that it is not a syphiloderm although, because of the high endemic syphilis and seropositivity rates in that area, the two conditions often co-exist in the same patients. 'Witkop' may, therefore, be regarded as a regional synonym for favus.

We gratefully acknowledge permission from the Regional Director (Africa) of WHO to use data compiled during the course of a WHO survey into endemic syphilis in the Bechuanaland Protectorate. We thank Prof. E. H. Cluver, Director of the South African Institute for Medical Research for his continued interest in the survey and Dr. L. J. A. Loewenthal for critically reading the script. The illustrations have been prepared by Mr. M. Ulrich of the Photographic Department of the S.A.I.M.R.

POSTSCRIPT

Since the completion of this work Scott and Kaplan[12] have reported a further case of witkop from whom *T. schoenleini* was isolated. The serological tests for syphilis were negative. They also concluded that witkop is a favus infection and not a syphiloderm.

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EDITORIAL

THE SURGERY OF ULCERATIVE COLITIS

Much has been written recently concerning ulcerative colitis, but the pathogenesis of this disease remains obscure.

The occurrence of lesions in the lower ileum in association with the condition has been recognized for many years, but there has been no unanimity of opinion with regard to their significance. It is generally agreed that the underlying mechanism of this retrograde ileitis is reflux of infected colonic contents through an ileo-caecal valve which has been rendered incompetent by the disease process, although in some cases the valve has been shown to function normally. McCready[1] has expressed the view that retrograde ileitis is a potent cause of mortality after ileostomy, and that since spread of the ileal inflammation could be anticipated, it was essential that operation should be performed above the level of ileal involvement. The other side of the coin is shown by Counsell[2] and by Brooke,[3] who take the view that, even when the ileostomy is made in a diseased segment of ileum, no harm will result and healing is to be expected and that in fact retrograde ileitis is not of very serious import at all.

Of much graver prognostic significance is the ileitis occurring in association with subacute obstruction of the ileostomy, and to which the term 'ileostomy dysfunction' has been applied by Warren and McKittrick.[4] Characteristically there is a watery discharge from the ileostomy, and abdominal colic, nausea and vomiting are usually present. It has been estimated that stenosis of the ileostomy occurs in about 40% of patients, the great majority of whom recover with conservative measures. In the presence of prolonged obstruction, however, ileal ulceration of a degree severer than that found in retrograde ileitis ultimately occurs,[2] with the very real risk of perforation and peritonitis. Stenosis of the ileostomy stoma has become decidedly less of a problem since Brooke[5] introduced his method of mucosal eversion with suture to the skin in 1952.

With regard to the medical management of ulcerative colitis, Brooke[6] while acknowledging the value of cortisone, has sounded a warning note concerning its indiscriminate use. He has shown that in patients who have not responded to cortisone over a period of 4-6 weeks, the colon may become extremely friable and even actively disintegrate, and so

VAN DIE REDAKSIE

DIE CHIRURGIE VAN ULSERERENDE DIKDERM-ONTSTEKING

Gedurende die afgelope tyd is heelwat oor ulsererende dikdermontsteking geskryf, maar die patogenese van hierdie siekte bly nog onduidelik.

Vir baie jare reeds word die voorkoms van letsels in die laer gedeelte van die kronkelderm by hierdie toestand besef, maar daar was geen eenstemmigheid van mening oor hulle betekenis nie. Daar is algemene ooreenstemming dat die onderliggende meganisme van hierdie terugkerende kronkeldermontsteking terugvloei van besmette dikderminhoud deur die dikdermklep, wat weens die siekteproses ondoeltreffend geword het, is, alhoewel dit getoon is dat die klep by sekere gevalle normaal funksioneer. McCready[1] het die mening uitgespreek dat terugwerkende kronkeldermontsteking ná ileostomie 'n ernstige sterfte-oorsaak is en dat, aangesien verspreiding van die kronkeldermontsteking verwag kan word, dit noodsaaklik is dat daar bo die gedeelte wat deur die ontsteking aangetas is, geopereer behoort te word. Die anderkant van die saak is deur Counsell[2] en Brooke[3] getoon, wat van mening is dat, al word die ileostomie in 'n aangetaste deel van die kronkelderm gedoen, dit nie skadelike gevolge sal hê nie en genesing kan verwag word, en dat terugwerkende kronkeldermontsteking inderdaad glad nie baie ernstig van aard is nie.

Van baie gewigtiger prognostiese belang is die kronkeldermontsteking wat by subakute verstopping van die ileostomie voorkom, en waarop Warren en McKittrick[4] die uitdrukking ‚abnormale werking van die ileostomie' toegepas het. Kenmerkend is daar 'n waterige afskeiding van die ileostomie, en buikkoliek, mislikheid en braking kom gewoonlik voor. Dit is geskat dat stenose van die ileostomie by ongeveer 40% pasiënte voorkom, waarvan die grootste meerderheid met behoudende behandeling herstel. Waar daar egter 'n langdurige verstopping aanwesig is, kom 'n ernstiger graad van kronkeldermulserering, as wat by terugwerkende kronkeldermontsteking aangetref word, uiteindelik voor,[2] met die baie wesenlike gevaar van perforasie en buikvliesontsteking. Stenose van die ileostomie-opening bied beslis nie meer so 'n groot probleem sedert Brooke[5] in 1952 sy metode van slymvliesomdopping met hegting aan die vel ingestel het nie.

Sover dit geneeskundige beheer van ulsererende dikdermontsteking aangaan, het Brooke,[6] terwyl hy die nut van kortisoon erken, gewaarsku teen die onverantwoordelike gebruik daarvan. Hy het getoon dat die dikderm uiters bros kan word en selfs aktief uitmekaar kan breek by pasiënte wat oor 'n tydperk van 4-6 weke nie op kortisoon reageer nie, en sodoende ‚die een kans van oorlewing wat die toevallige pasiënt deur kolektomie kan verkry, benadeel'. Hy raai

'prejudice for the occasional patient the one opportunity of survival which can be obtained through colectomy'. He therefore advises that steroid therapy should cease in the absence of a response within 2 weeks.

The indications for surgery are now well established. They include fulminating cases, continued deterioration despite medical measures, acute emergencies such as haemorrhage and perforation, pseudopolyposis, malignant degeneration and, finally, systemic manifestations such as arthritis, iritis, and pyodermia. It has been estimated that about 30-40% of all cases will eventually come to surgery, which should aim at excision of the diseased colon and rectum with the institution of a permanent ileostomy.

The operative programme may involve a staged procedure or a one-stage panproctocolectomy, with the present-day trend towards the latter operation. There is general agreement that ileostomy alone is inadequate since the continual toxic absorption together with the loss of blood and protein into the lumen of the diseased bowel and the ever-present danger of perforation may lead to a fatal outcome. 'These colons are in effect nothing more than a serosal sac filled with faeces, pus and blood and lined only with shaggy ulcers. The physiological effect of such a colon is comparable to that of an extensive third-degree burn covered with a faecal poultice.'[7] Agreement is less general on the wisdom of performing total colectomy with ileo-rectal anastomosis. The temptation to leave the patient with a functioning sphincter apparatus is great, but it should be remembered that the rectum is free from disease in only about 8% of cases, and that there is a definite risk of malignant degeneration occurring in the rectal stump.[8] Excoriation of the perianal skin from the eroding effects of ileal contents is also a potential hazard of this procedure, as are other complications, which include stenosis of the stoma and recto-vesical fistula.

Aylett,[9] on the other hand, has long been convinced that preservation of the rectum is possible in nearly all cases. He has recently reported on 47 consecutive cases so treated and, on the basis of rectal biopsies taken before and after operation, has established that after total colectomy, resolution of the rectal inflammation is to be expected, although the normal histological pattern of the rectal mucosa is not entirely regained. An analysis of his results reveals that 70% of his patients have been able to return to their normal social activities, and a further 15% have only minor limitations due to some frequency of bowel action. In none of the cases has conversion to ileostomy been required. On the basis of these results Aylett concludes that total colectomy with ileo-rectal anastomosis is the procedure of choice in the surgery of ulcerative colitis. There can be no doubting the importance of the Aylett experiment, but a much longer follow-up is needed (the series began in 1951) before final judgment can be pronounced on the merits of the procedure.

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derhalwe aan dat, indien daar binne 2 weke geen reaksie is nie, steroïed-terapie gestaak behoort te word.

Die aanduidings vir chirurgie is nou behoorlik vasgestel. Hulle omvat uitbarstende gevalle, aanhoudende agteruitgang ten spyte van geneeskundige maatreëls, akute noodgevalle soos byvoorbeeld bloeding en perforasie, pseudopolipose, kwaadaardige degenerasie en ten laaste, sistemiese tekens soos byvoorbeeld gewrigsontsteking, reenboogvliesontsteking en etterende huidontsteking. Dit is geskat dat ongeveer 30-40% van alle gevalle mettertyd chirurgie moet ondergaan wat op verwydering van die besmette dikderm en rektum met die instelling van 'n permanente ileostomie gemik behoort te word.

Die operasie kan in stappe gedoen word of 'n panproktokolektomie, wat slegs een operasie verg, kan gedoen word. Laasgenoemde operasie vind hedendaags groter byval. Dit word algemeen ooreengestem dat ileostomie op sigself ondoeltreffend is, aangesien die aanhoudende toksiese absorbering, tesame met die verlies van bloed en proteïen in die binneruimte van die besmette dikderm en die altydaanwesige gevaar van perforasie, noodlottig mag wees. ‚Hierdie dikderms is in werklikheid net 'n sak van weivlies, gevul met ontlasting, etter en bloed, en oortrek met ruie ulsers. Die fisiologiese uitwerking van so 'n dikderm kan met dié van 'n derde-graadse brandwond, wat met 'n pap van warm ontlasting bedek is, vergelyk word.'[7] Daar bestaan nie dieselfde mate van ooreenstemming oor die raadsaamheid om algehele kolektomie met ileo-rektale anastomose uit te voer nie. Die versocking om die pasiënt met 'n werkende sfinkterstelsel te laat is groot, maar dit moet in gedagte gehou word dat die rektum slegs by ongeveer 8% gevalle nie besmet is nie, en dat daar 'n definitiewe gevaar is dat kwaadaardige degenerasie in die rektale oorblyfsel[8] sal voorkom. Afskawing van die perianale vel deur die vretende werking van kronkelderminhoud is ook 'n wesenlike gevaar by hierdie metode, soos ook ander komplikasies wat stenose van die opening en rekto-vesikale fistel insluit.

Aan die anderkant was Aylett[9] lank reeds oortuig dat behouding van die rektum by byna alle gevalle moontlik is. Hy het onlangs verslag gelewer oor 47 opeenvolgende gevalle wat op hierdie wyse behandel is, en op die grondslag van rektale biopsies wat voor en na die operasie gedoen is, het hy vasgestel dat opklaring van die rektale ontsteking na algehele kolektomie verwag kan word, alhoewel die normale histologiese voorkoms van die rektale slymvlies nie heeltemal dieselfde sal wees nie. Ontleding van sy resultate bring aan die lig dat 70% van sy pasiënte in staat was om na hulle normale sosiale werksaamhede terug te keer, en dat 'n verdere 15% slegs tot 'n mindere mate aan bande gelê is weens taamlik dikwelse stoelgang. By nie een van hierdie gevalle was dit nodig om tot ileostomie oor te gaan nie. Op die grondslag van hierdie resultate het Aylett tot die gevolgtrekking gekom dat algehele kolektomie met ileo-rektale anastomose die uitverkore chirurgiese metode vir ulsererende dikdermontsteking is. Daar kan geen twyfel bestaan oor die belangrikheid van die Aylett-proefneming nie, maar dit moet oor 'n langer tydperk strek (die reeks het in 1951 begin) voordat die finale uitspraak oor die verdienstelikhede van die metode gelewer kan word.

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CORONARY DISEASE AND DIET

In an article in this issue of the *Journal* (p. 663) Brock and Gordon summarize their views concerning the effect of diet on coronary disease. They refer to the voluminous and sometimes confusing literature on the subject, and submit a balanced account of current concepts on the dietary factor in this disease with a view to dispelling some of the confusion that exists. While they do not fail to point out that diet is only one of several factors concerned in atherogenesis, they hold that no factor is more important or easier to modify.

Starting from the *assumption* (and they put it no higher) that the cholesterol content of the blood is an index of liability to coronary attacks, our authors analyse the factors which increase the serum-cholesterol level and proceed to explore measures which will reduce this level. They find, generally speaking, that a diet rich in animal fats and hydrogenated oils, that is to say, in saturated fatty acids, raises the cholesterol level; whereas oils of vegetable and marine origin, rich in unsaturated fatty radicles, do not have this effect and may, in fact, actually depress the cholesterol level.

On this basis tentative dietary advice is offered for the management of acute cardiac infarction and for the prevention of this form of heart disease in persons particularly at risk and in the privileged communities as a whole. While the effects of such dietary measures will only be finally assessed after a long period of investigation, these measures offer to the patient who has survived a coronary attack the satisfaction of doing something to prevent a recurrence and, to other people in the groups particularly at risk, positive measures to safeguard themselves against cardiac trouble.

We wrote on this subject last year in our editorial columns,[1] and it was then stressed that the startling results that had been obtained in the investigation of the connection between dietary fat and coronary disease should be interpreted with caution, and that we should beware of overmuch dogmatism in disseminating advice on the subject of diet. The same cautious note is now struck again by Brock and Gordon, who conclude their article by emphasizing that the more definite advice they now give is still tentative and subject to modification in the light of further knowledge.

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THE DIETETICS OF CORONARY HEART DISEASE

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Coronary heart disease is the commonest cause of death in civilized communities to-day and therefore presents a great challenge to modern preventive and therapeutic medicine. The steadily increasing incidence in coronary deaths in the European population of South Africa is illustrated in Table I.

TABLE I. THE RISING INCIDENCE OF DEATHS FROM CORONARY HEART DISEASE IN THE EUROPEAN POPULATION OF THE UNION OF SOUTH AFRICA

(1940–1954)

Year	*Total Population*	*Total Deaths*	*Deaths from Coronary Heart Disease*
1940	2,161,000	20,277	1,240
1950	2,610,000	22,717	3,062
1954	2,803,000	24,106	3,929

This table, and Fig. 1 have been compiled from data kindly provided by the Director of Statistics, Pretoria.

Part of this increase may reflect increased awareness of the disease picture and improved diagnostic technique; but by 1940 the various clinical syndromes of coronary atherosclerosis had long been clearly delineated and were widely known and it is inconceivable that the striking increase in the certification of coronary deaths during these 14 years is due only to better diagnosis; it must surely indicate a true increase in the disease incidence.

It has been customary to classify coronary heart disease among the 'degenerative diseases' and to regard coronary atherosclerosis along with grey hair, presbyopia and failing memory as an inevitable consequence of aging. But many cases of this disease are being diagnosed in men in the prime of their lives and the data in Fig. 1 reveal that in contemporary medical practice coronary heart disease is a major problem in young adult and middle-aged men and not only in the older age-groups.

Fig. 1. The age distribution of deaths from coronary heart disease in the European male population of South Africa in 1954.

The present approach to the management of the disease is based on such epidemiological observations. While it may be true that an inherent defect in lipid metabolism and other genetic factors play uncontrollable roles in the development of coronary atherosclerosis, the annually increasing incidence of the disease indicates the importance of environmental influences in its pathogenesis. The disease is now regarded as an 'epidemic of modern civilization'[1] and like all epidemics may prove to be successfully controlled by eradicating its environmental causes. Acceptance of the hypothesis that coronary heart disease is not an inexorable degenerative process has given a considerable impetus to the search for agents which will inhibit its course.

A voluminous literature bears testimony to the intensity with which world-wide research into these problems has been conducted. Often, enthusiasm has broken the bounds of adequate clinical control and the practitioner who is daily concerned with the management of coronary heart disease must be considerably perplexed by the mass of often contradictory data with which he is presented. In the hope of dispelling some of the existing confusion, we are attempting here to provide a balanced account of current concepts of the dietary factor in this disease. This is but one of several factors concerned in atherogenesis. Apart from genetic factors, the endocrine secretions of the thyroid, gonad, adrenal and pancreas have been implicated and the possible significance of exercise, emotion and smoking must not be overlooked. At present, however, none of these appears to be more important or easier to modify than the diet, which therefore merits our closest attention.

By way of clarification, it must be pointed out that in short-term human studies, direct observation of the clinical or morbid anatomical course of the disease process is not feasible and most therapeutic trials are based on the *assumption* that a direct relationship exists between the level of the blood lipids and the development of atherosclerosis. It is further *assumed* that a reduction in these levels will produce a beneficial change in the course of the disease. Of the many modalities of lipid biochemistry which can be studied, we have selected the serum cholesterol. It may not be the measurement which correlates best with the clinical or pathological state but it has the merit of being simple to determine and is readily available in routine laboratory services. It should be noted, however, that the blood lipids (including the serum cholesterol) have dynamic functions and vary considerably in response to many known and unknown changes in the internal and external environment. Accordingly, isolated determinations of the serum cholesterol are of little significance and serial readings for long periods before, during and after withdrawal of treatment are essential when evaluating the efficacy of a therapeutic regime.

A. Dietary Factors in the Aetiology of Coronary Heart Disease

1. *Calories and Obesity*

It is widely assumed that the obese are specially prone to coronary heart diesease. Even Hippocrates[2] has recorded that 'sudden death is more common in those who are naturally fat than in the lean'. Modern studies, however, have failed to demonstrate a causal relationship between obesity and coronary heart disease. Our own limited metabolic experiments have shown that when obesity is produced by long-term feeding of high carbohydrate diets, providing up to 4,500 calories, very little rise in blood cholesterol is produced. More directly, a report from the United States Army[3] revealed that weight distribution at enlistment of 233 men who subsequently died of necropsy-proven coronary heart disease was the same as in those who remained clinically well. In a classical investigation,[4] Gertler and White found that the weights of 100 young people with coronary heart disease did not differ materially from those of healthy controls. In fact, the incidence of the disease in the plump 'endomorph' was substantially less than in the stocky, muscular 'mesomorph'. And in the South African Bantu corpulence is not rare but the incidence of coronary heart disease is negligible.[5]

Nevertheless, insurance-company statistics often reveal an increased mortality from coronary heart disease in 'overweight' policy holders.[6] In evaluating insurance statistics it must be borne in mind that 'overweight' is not the same as 'obesity'; the latter implies an excess deposition of fat, while the former indicates a deviation above the mean for that age, sex and height. Furthermore, the available data do not clearly separate diabetics from the over-all group of coronary deaths. Finally, the increased incidence of coronary deaths in the 'overweight' group does not necessarily mean that there is a causal relationship and may merely represent the deleterious effect of obesity on the course of established heart disease in general. So although obesity *per se* is not an important factor in the aetiology of coronary heart disease, considerable attention must be paid to its correction in the management of the established disease.

2. *Quantity of Dietary Fat*

Of all the environmental variables studied, none correlates so well with incidence of coronary heart disease as the level of fat in the diet. This important association was initially demonstrated in 1952 by the epidemiological studies of Ancel Keys[7] and it has since been extended by field surveys in many parts of the world.[8] In general, the data reveal that in populations consuming little fat, the incidence of coronary

heart disease is low (e.g. in Japan 9% of the calorie intake is derived from fat) while in those consuming large amounts of fat (USA—40% fat calories) the incidence of coronary heart disease is high. In a given race, differences in the incidence of coronary heart disease in various sections of the population may be correlated with the fat intake; thus in Italy the incidence of coronary heart disease is much higher in Bologna (29% fat calories) than in Naples (20% fat calories).[9] This relationship was particularly well seen in the Cape Peninsula, where the incidence of coronary heart disease and the height of the serum cholesterol in the local racial groups correlates in striking fashion with the fat content of the diet.[5] The Bantu obtains only 17% of his calories from fat and has a low serum-cholesterol level and very little coronary heart disease; 35-40% of the European's calories are from fat, and he has a relatively high serum-cholesterol level and incidence of coronary heart disease; the Cape Coloured is intermediate in his fat intake (25% fat calories), has an intermediate level of serum cholesterol and an intermediate susceptibility to coronary heart disease.

Under our experimental conditions, in those patients whose serum-cholesterol levels were only slightly elevated by carbohydrate feeding, calorically equivalent or smaller amounts of animal or coconut fat promptly produced sharp rises in the serum-cholesterol level. Clearly, then, very close attention must be paid to the quality of fat in the diet in the management of coronary heart disease.

3. *Quality of Dietary Fat*

Although the general principle 'high-fat diet, high incidence of coronary heart disease' is widely applicable, recently interest has been focussed on possible *qualitative* differences in the cholesterogenic properties of fats. In Holland, Groen *et al.*,[10] in 1952, varied the diets of 60 normal subjects and found that serum-cholesterol levels were lower when predominantly vegetable diets were given than when the diets were rich in animal fats.[10] Both in the Netherlands[11] and in the USA[12] surveys showed that pure vegetarians had substantially lower serum cholesterols than non-vegetarians and in the inter-racial survey in the Cape Peninsula[5] a correlation between fat consumption and serum-cholesterol level only existed for animal fat and not for the vegetable fat intake.

Feeding experiments in several parts of the world have confirmed the differential action of various fats on the serum cholesterol.[13-18] The results of these experiments may be summarized as follows:

1. Fats differ in their effect on the serum-cholesterol; some raise its level, some lower it, and some have no effect.

2. The fats which lower the serum cholesterol are generally from vegetable sources (e.g. sunflower seed, cotton seed, corn, soybean) or of marine origin (e.g. pilchard, seal, sardine). These fats are generally liquid at room temperature (i.e. they are oils) and contain large amounts of 'essential fatty acids' (linoleic, linolenic, arachidonic, pentaenoic and hexaenoic acids). These oils are highly unsaturated, as indicated by their iodine values, which are generally over 100.

3. Not all vegetable fats behave similarly. Some less unsaturated oils (iodine value 80-90) with lesser amounts of essential fatty acids, do not depress the serum cholesterol significantly; olive oil and peanut oil represent this category. Some vegetable fats behave like animal fats and raise the serum cholesterol; coconut fat (iodine value 9) is typical of this group.

4. When liquid vegetable oils are artificially hardened ('hydrogenated'), as in the manufacture of margarine and certain cooking fats, they lose their cholesterol-lowering properties and behave like animal fats. Like animal fats (lard, butter, dripping), they have iodine values below 50 and practically no essential fatty acids and they promptly raise the serum cholesterol of the human subjects to whom they are given.

5. The effects of the various fats are independent of changes in body weight and calorie intake and of the protein and vitamin content of the diet.

The qualitative differences of these fats have hitherto been studied in short-term experiments only, but trials over extended periods are at present being conducted in several institutions, including our own. It is our preliminary impression that, even after allowing for considerable spontaneous fluctuation, low serum-cholesterol levels can be maintained for periods of up to 6 months by feeding with certain oils. (Fig. 2).

Of particular interest is the observation of the 'neutralizing' effect of one type of fat on another. Thus we have been able to counter completely the cholesterol-raising effect of 100 g. of hydrogenated coconut oil by simultaneously giving 100 g. of sunflower-seed oil, even though the fats in this diet provided 1,800 out of a total of 4,000 calories; i.e. a fat/calorie ratio of 45%.

The mechanism of action of the different fats is at present unknown but is the subject of considerable research. Nor is it clearly established which component of the natural oils is reponsible for the cholesterol-lowering effect. It may be a function of unsaturation in general, or of the essential fatty acids only. Or it may not be mediated by the fatty acids at all and may reside in the unsaponifiable portion of the oil.

Much research on the role of these fats in atherogenesis is still needed and the available data must be cautiously evaluated. Nevertheless, it is clear that not only quantity but also quality of fat must be taken into account when planning the diets of patients with coronary heart disease.

4. *Protein*

In some early experimental work on rabbits it was shown that heavy protein feeding produced atherosclerosis.[19] These experiments need to be interpreted cautiously because the cholesterol content of the high-protein feeds was not always controlled and the rabbit is particularly prone to atheromatosis from dietary cholesterol. On the other hand, a diet deficient in sulphur-containing amino acids (together with a high fat and cholesterol intake) is used experimentally in the production of atherosclerosis in monkeys.[20] The correlation of these observations with human affairs is not clear. The Bantu diet is notoriously poor in protein and they do not suffer from coronary heart disease.

Observations on our metabolic subjects fail to demonstrate any effect on the serum cholesterol of a changing level of protein intake[15] and recently, a more detailed study by Keys and his associates has confirmed this.[21]

5. *Dietary Cholesterol*

The average European diet contains less than a gram of cholesterol per day. This small intake contributes very

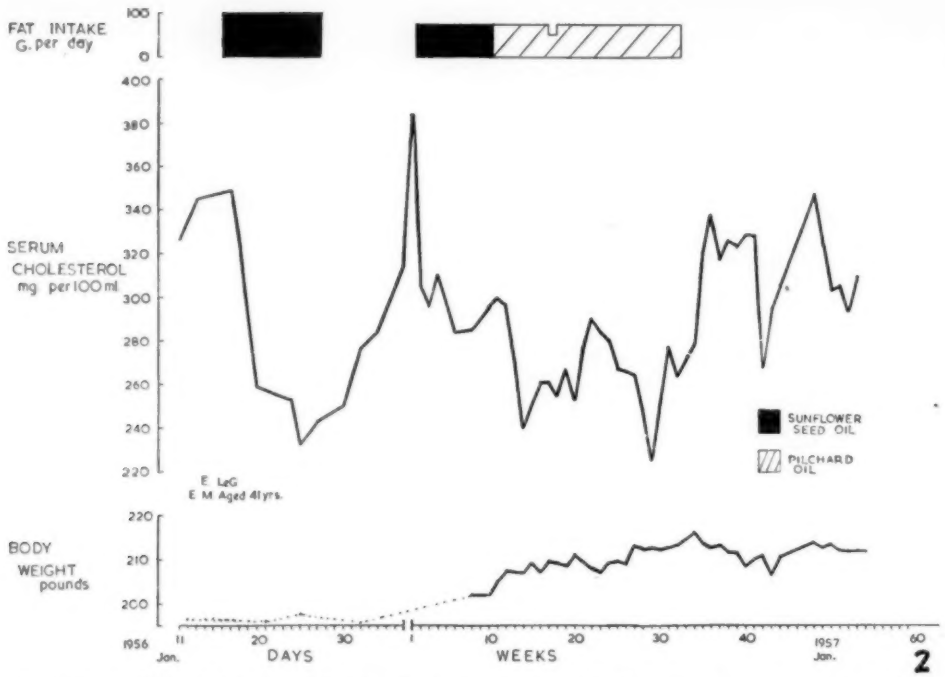

Fig. 2. The variations in the serum cholesterol of a European man during more than a year's observation. The sustained reduction in the serum cholesterol during eight months of supplementation of the diet with sunflower seed or pilchard oil is clearly demonstrated. Nevertheless, unexplained fluctuations in serum cholesterol level from week to week are also present. Note that while on the fat supplement, weight was gained while the serum cholesterol fell. This weight gain was allowed for experimental purposes.

little to the total cholesterol in the body, most of which is synthesized endogenously. Nevertheless much attention has been paid to 'low-cholesterol' diets in the management of coronary heart disease. This misconception has arisen in two ways: Firstly, in animal experiments, cholesterol feeding produces hypercholesterolaemia and atheromatosis. In these experiments, however, highly artificial diets are employed containing very large amounts of cholesterol, which on a proportionate body-weight basis are grossly in excess of those in the most liberal human diet. Secondly, confusion has arisen because most 'high-cholesterol' foodstuffs (e.g. eggs, meat, butter) also contain large amounts of fat and the effect on the blood lipids of these diets is a function of their fat content and not of the cholesterol. This was shown by Messenger *et al.*,[22] who fed 5 subjects with 30 g. of cholesterol (the equivalent of 100 eggs) daily with very little effect on their serum-cholesterol levels. When cream or egg-yolk fat was added to the diet, the blood cholesterol rose sharply. Keys *et al.* showed that 10 g. of cholesterol in a single meal only produced a trivial rise in serum cholesterol[23] and in a series of dietary surveys found no correlation between the dietary cholesterol and its blood level.[24] In our Department, Bronte-Stewart *et al.*[15] confirmed that large doses (3 g.) of cholesterol produced only small rises in the serum cholesterol when compared with certain fats.

It is concluded, therefore, that for practical purposes the cholesterol content of the diet need not be considered in the management of coronary heart disease.

6. *Cellulose*

Vegetarian diets in privileged communities and the primitive diets of underprivileged people are known to be high in cellulose. Thus, a feature of the Bantu diet is its high content of coarse cereal products and its ingestion is accompanied by a high faecal excretion of fat and sterol. Walker has suggested that such a diet, in conjunction with a low fat intake, may be a factor in protecting the Bantu from coronary heart disease.[25] This is a most interesting suggestion and is worthy of further epidemiological and experimental investigation, but at present there is insufficient evidence to justify raising the cellulose content of the diet in the management of coronary heart disease.

7. *Vitamins*

Many of the vitamins play essential roles in the intermediate metabolism of the lipids but despite a fair amount of experimental work no clear-cut relationship between the vitamin supply and human atherosclerosis has been demonstrated.

(*a*) *Pyridoxine.* Pyridoxine-deficient monkeys develop intimal damage and fibrosis[26] and they are also more susceptible than control monkeys to diet-induced hypercholesterolaemia.[27] It should be emphasized, however, that these pyridoxine-deficient, hypercholesterolaemic monkeys

do not develop true atherosclerosis. Nevertheless, because of these observations and because pyridoxine is known to promote the synthesis of the essential fatty acid, arachidonic acid, from linoleic acid,[28] clinical trials have been made with this vitamin.[29] The experimental regimen consisted of a diet low in total fat, but relatively high in unsaturated fat, and was supplemented by both vitamin B_6 and by calcium disodium versenate. Lowering of the serum cholesterol was obtained but the particular contribution of the vitamin B_6 to this result cannot be assessed.

(*b*) *Tocopherol.* Vitamin E has been recommended for the treatment of angina pectoris[30] but its value in this condition has not been satisfactorily demonstrated. Le Riche[31] has drawn attention to the large amounts of tocopherol in vegetable oils and asks 'whether the cholesterol was lowered by the essential fatty acids or by the tocopherols in the Cape Town experiments?' In these experiments, however, marine oils which are particularly poor in tocopherol content often produced greater reduction in the serum-cholesterol level than the vegetable oils.[15] Human diets are generally adequate in vitamin-E content and it is unlikely that simple deficiency of this vitamin is concerned with the aetiology of coronary heart disease.

(*c*) *Nicotinic Acid.* Two groups of workers have reported 'pilot experiments' in which reduction in the serum-cholesterol level was obtained by the administration of 3 g. of nicotinic acid daily.[32, 33] Neither report mentions any control of the subjects' diet or weight and in neither investigation was there an adequate control period before and after therapy. The rationale of this method is obscure. It is clearly not a physiological effect of the vitamin because nicotinamide administration did not reproduce the effect of nicotinic acid. Nicotinic acid has of course been used in the past in angina pectoris for its pharmacological vaso-dilator effect; it may produce unpleasant flushing and sometimes nausea and unless its therapeutic efficacy is more convincingly demonstrated it is hardly likely to become an acceptable form of long-term treatment.

(*d*) *Vitamin D.* Excess vitamin D potentiates the development of atherosclerotic lesion in choline-deficient rats[34] but the importance of this phenomenon in human atherogenesis is not known.

8. *Sitosterols*

Phytosterols are a large group of naturally occurring substances found in higher plants. Certain members of this group, the sitosterols have a very similar chemical structure to cholesterol and are able to inhibit the absorption of dietary cholesterol and may also block the re-absorption of excreted endogenous cholesterol.[35] Beta-sitosterol has been subjected to many clinical trials[36-38] and when given in large, unnatural doses (20-25 g. daily), 10-15% reduction in serum-cholesterol level has been achieved. Again, the clinical trials must be criticized on the grounds that the subjects' diet and weight were not adequately controlled. The large doses of sitosterol are not pleasant to take and in their present form are not suitable for the long-term prevention and management of coronary heart disease.

9. *Lipotropic Agents*

The lipotropic agents (particularly choline and inositol) have been widely advertised for their value in the management of atherosclerosis. A perusal of the literature, however, reveals that the reputation of these substances stands on flimsy theoretical, experimental and clinical foundations. Theoretically, it is hoped that lipotropic agents will clear fat from atheromatous arteries in the same way as they are said to clear fatty livers; but both biochemically and histologically, atheroma and 'fatty liver' differ vastly and cannot be expected to behave similarly. The reported experimental data are extremely contradictory but the most recent and most careful investigations have failed to show any beneficial effect of choline in the prevention or regression of cholesterol induced atherosclerosis in rabbits,[39] chickens[40] and dogs.[41] In the rat, however, choline deficiency may produce arterial damage, and this may be further aggravated by cholesterol feeding.[42] The only large-scale clinical trial which has been reported is that of Morrison,[43] but evaluation is not possible because, apart from lipotropes, a low-fat diet was also given, and weight changes were not checked.

10. *Alcohol*

The role of alcohol in atherogenesis is uncertain. In a small survey of elderly men, Hobson *et al.*[44] found an appreciably lower serum cholesterol in 12 heavy drinkers (224 mg.%) than in 14 teetotallers (305 mg.%), and in an autopsy series Wilens[45] found less atherosclerosis in alcoholics than in the general population. In both these series, however, variables other than alcohol were present, and we conclude with Page[46] that 'there is no substantial evidence that alcohol aids in atherogenesis or that it dissolves the lesions once they are present except in concentrations used in histological preparations!'

B. Practical Dietary Advice on the Management and Prevention of Coronary Heart Disease

The large body of epidemiological, clinical and experimental evidence leaves little doubt that dietary fat is somehow implicated in the pathogenesis of coronary heart disease. But when it comes to laying down rules for the practical management of the disease it must be admitted at once that the evidence is by no means complete and is sometimes contradictory.

Those practitioners who insist that their therapy must be based on procedures which have been established by controlled clinical trial, or which have withstood the test of time, will not be satisfied with the available evidence and will prefer to wait for further developments. Others, however, will accept that dietary modification of the disease is possible; in other words, they will be prepared to act on a reasonable hypothesis. We agree that such an attitude is justifiable, provided that the practitioner is frank with himself and with his patients about the incompleteness of the evidence on which dietary therapy in coronary heart disease is based.

Against this background, we shall deal with the problem (1) as it relates to the prevention of coronary heart disease in predisposed persons, (2) as it affects the management of the established disease, and (3) as it concerns the dietary advice to privileged communities in general.

1. The Prevention of Coronary Heart Disease in the 'Population at Risk'

We cannot precisely define the 'population at risk'. Some would say that in privileged communities all men and, at

least, the post-menopausal women are 'at risk'. Most people will agree, however, that this category must include patients with symptoms of coronary disease and those who have made a partial or complete recovery from an attack of cardiac infarction. Diabetics, and men with a family history of coronary disease, are also included; those of a mesomorphic habitus are particularly prone. In the present state of knowledge, the most practical dietary advice which can be offered to this 'population' is that they reduce their fat intake.

Many clinical studies of the effect of fat restriction are currently in progress but so far very little has been published and only two series of cases are available for discussion. The first is that of Morrison,[43] who studied 100 cases of cardiac infarction, half of whom were allowed to eat freely and who consumed an estimated 80-160 g. of fat daily. The other half were put on a strict low-fat diet providing 1,600 calories daily, of which about 16% were derived from fat (20-25 g. of fat daily). In addition the latter group were given large amounts of whole wheat germ, and brewers' yeast daily. After 8 years of follow-up, 12 of the 50 controls were still alive; of the 38 deaths, 22 were from 'cardiovascular' causes. In the treated series, 28 were still alive and only 9 of the 22 deaths were attributed to 'cardiovascular' causes. The treated patients lost, on the average, about 15% of their body-weight on the above regime. Little is known about the diets of the controls and their weight changes are not recorded.

Lyons and his colleagues,[47] in California, studied 280 patients with cardiac infarction for an average period of 4 years. Of these, 155 were put on a low-fat diet; this allowed a maximum of 50 g. of fat a day, but the total calorie content of the diet was not stated. The remaining 125 consumed a free diet. The dieters maintained lower lipoprotein levels than the non-dieters; 15 episodes of recurrent cardiac infarction with 4 deaths occurred in the dieters, 51 recurrences occurred in the non-dieters; with 13 deaths.

These two studies are not complete, and certainly not conclusive; nevertheless, their findings are consistent with the epidemiological data and support our recommendation to the population at risk to restrict their fat intake. Such restriction cannot be undertaken lightly. To achieve a diet providing only 20% of its calories from fat it is necessary to reduce to a minimum all meats (with the possible exception of breast of chicken, tripe, kidneys and liver), dairy products (including milk, butter, cheese, cream, eggs and ice-cream, but excepting skim milk), fried foods, pastries, chocolates, and salad dressings. Fats have the important function of making the diet attractive and palatable and they have the advantage of providing calories without undue bulk. The continued consumption of a strict low-fat diet will quickly become irksome and may produce a considerable psychological disturbance in the patient (and often in his wife!) with the development of irritability and mental depression.[48] In addition there may be gastro-intestinal disorders with diarrhoea or constipation and considerable flatulence.[46]

Hence only moderate restriction of dietary fat is practicable and it is unlikely that reduction of the average intake to less than 30% fat calories will be generally acceptable; on such a regime a substantial reduction in serum cholesterol cannot be anticipated.[49] Some of the recent advances in the dietetics of coronary heart disease may possibly improve the patients' lot. Very recently, Wilkinson[50] drew attention to the observation that in idiopathic hyperlipaemia, a rare inborn error of lipid metabolism which is often associated with hypercholesterolaemia and coronary heart disease, the essential biochemical lesion is the body's failure to clear lipid from the plasma. Thus the postprandial lipaemia, which is normally cleared in 5-6 hours, may persist for up to 24 hours and each meal produces a cumulative increase in blood fat. Wilkinson accordingly suggests that in patients with this disease a single high-fat meal a day is quite in order provided that the other two meals are virtually fat-free; the body will then have time to clear the plasma of lipid during the 24 hours between meals. Now, there is evidence that in coronary heart disease, a similar defect of lipaemia clearance exists[51] and one can speculate on the possibility of managing coronary heart disease by prescribing a fat-free breakfast and lunch but allowing the patient to indulge in one fatty meal at night. This is at present only a theory, and cannot be recommended for general application without much further experimental investigation.

On somewhat less speculative grounds is the possibility that restriction need only be applied to saturated fats which raise the serum cholesterol and that a substantial intake of natural vegetable and marine oils may be permitted or even encouraged. On this principle, only moderate reduction in animal fat and dairy products is required and the diet is made more palatable by the liberal use of oils for frying, salad dressings, puddings, pastries etc. Such diets will necessitate greater use of fish, fruit and vegetables at the expense of meat, dairy products and hydrogenated fats; but, with a minimum of culinary ingenuity, tasty meals should be easy to prepare. All pastries, except 'flaky pastry', can be reasonably made with oil instead of fat; fish can be fried in its own oils rather than in butter; and the standard breakfast of bacon and eggs can be supplanted by a cereal with a little milk, by a fried kipper or by compote or fresh fruit.

There is little published data on the value of such diets in practice. Schroeder[29] has reported on 20 patients, with and without atherosclerosis and with normal or high initial blood-cholesterol levels. After a short control period in which up to 6 determinations of the serum cholesterol were made, the patients were put on a complicated regimen comprising a diet low in saturated but high in unsaturated fats, with supplements of soybean oil, pyridoxine and calcium disodium versenate. The serum cholesterol of most of the subjects fell, often substantially. After 4 months the mean fall for the group was 20% (i.e. from a mean of 257 mg.% to 200 mg.%). No check was kept of the patients' weights. The regimen includes several variables, and so it is not possible to assess the precise role of any one of them in reducing the serum cholesterol.

More recently, van Handel *et al.*,[38] from Holland, reported that they had to abandon their low-fat diets and have investigated a diet in which lard and other animal fats, shortening, white bread and degerminated cereals were banned; margarine and butter were restricted to 25 g. daily; whole-wheat bread, soybean oil, lean meat, milk, cheese and eggs were allowed *ad lib.* The diet included an obligatory supplement of 2-3 g. of soy lecithin, 25-50 g.

each of peanuts, peas or beans, and whole soybean flour. This complicated regimen, whose total calorie and fat content is not stated, was applied to 34 atherosclerotic patients for a mean period of 110 days. Adequate control data are not included in the report, but it is stated that substantial falls in serum cholesterol were obtained in those with high pre-treatment levels, whereas little or no fall was produced in those whose initial levels were low.

It must be emphasized that, apart from a large number of acute metabolic experiments, the above two inadequate clinical trials constitute the only published data on the value of unsaturated fats in the management of coronary heart disease. It is too early, therefore, unreservedly to recommend major changes in conventional eating habits and it is only as a concession to those who cannot tolerate a major reduction in their total dietary fat or in whom such a reduction does not produce the desired effect on the serum cholesterol, that a more liberal intake of unsaturated fats should be permitted.

2. THE DIETARY TREATMENT OF ESTABLISHED CORONARY HEART DISEASE

Semi-starvation in the Acute Stage

Dietetic principles have to be applied very early in the management of coronary heart disease. For the first few days after a cardiac infarction, severe dietary restriction should be advised, and Master *et al.*[52] claim that such a regime practically halves the mortality from acute coronary disease. Such a diet will depress body metabolism and this is valuable when the cardiac output is low. Epigastric distension and pressure on a tender congested liver are prevented, and restriction of bowel actions will not harm a shocked patient. Small fluid meals—fruit juices and jelly—are all that is needed until the patient is over the acute stage.

Fat Restriction

Later, the diet may be increased and at this stage, apart from consideration of salt restriction if heart failure is present, special attention must be given to the dietary fat. Several workers have presented evidence to show that lipid ingestion increases the coagulability of the blood.[53, 54] Although this evidence has not been universally accepted,[55] it is at least a theoretical indication for restricting the fat intake at this stage of the disease when extension of the lesion and thrombo-embolism are so likely to occur.*

Kuo and Joyner[56] have given a further reason for the restriction of dietary fat at this stage and, in fact, at any stage of the disease. These workers were able to induce angina pectoris and 'ischaemic' changes in the electrocardiograph by giving fatty meals. This may be the explanation of the greater liability of pateints with coronary heart disease to develop angina after meals.

When the patient is up and about (either with some residual disability or fully rehabilitated), steps must be taken to prevent recurrence of the disease and, if possible, to secure reversion of the lesions which are already present. The dietetic factors concerned with prevention of spread of the disease are discussed above. The same principles should be applied in an attempt to reverse the existing lesions. Atherosclerotic lesions are assumed to be reversible, both on experimental and on pathological grounds. Experimentally,[40] cholesterol-induced atherosclerosis in animals can be reversed by withdrawing cholesterol or by lowering the serum cholesterol (e.g. with oestrogens, sitosterol, etc.). Pathologically it has been observed that atherosclerosis is less marked at necropsy in those dying from wasting diseases than in the general population.[45] This might be interpreted as meaning that as a result of the wasting process regression of the lesions has occurred. It might, however, have other meanings.

Although therefore it can be demonstrated that the lesion in animals are potentially reversible, none of the available therapeutic regimens has yet been shown to produce such an effect in man.

Weight Reduction

There is little doubt that the prognosis of heart disease in general can be improved by reduction of weight to 'normal' or even less. Details of weight-reducing regimens are beyond the scope of this paper, but we would make the following observations:

(i) In the design of a weight-reducing diet, calorie reduction should as far as possible be achieved at the expense of the fat intake.

(ii) Many weight-reducing diets are irksome because they lack bulk. Apart from satisfying the patient, bulky diets containing large amounts of cereals, squash, pumpkin etc. have a possible theoretical advantage in their high cellulose content.

(iii) Considerable weight loss can be achieved by small dietary restrictions.[57] This can be shown by a simple example. A pound of body-weight is equal to about 3,500 calories and a slice of bread provides about 100 calories. Thus by cutting out one slice of bread a day 36,500 calories a year are saved and this represents a weight loss of 10 lb. Unfortunately, the converse also holds true, and even occasional small snacks between meals may add up to a considerable gain in weight.

(iv) Exercise has some value in weight reduction and an average man walking a mile loses 100 calories;[57] over a year this adds up to the calorie equivalent of 10 lb. of body-weight and is comparable to the sacrifice of a daily slice of bread. Many physicians hesitate to recommend exercise to cardiac patients, but strenuous activity is not necessary. A slowly ambled mile produces roughly the same calorie loss as a brisk walk over the same distance and, apart from putting less strain on the heart, does not stimulate the appetite unduly. There is much to be said for the cardiac patient living up to his effort tolerance. Exercise within his limits improves muscle tone; hence the mechanical efficiency of his body is increased and the load on his heart is reduced.

Psychological Management

It should be made clear to the patient that the dietary recommendations are only a part of his general management, and he should be prevented from becoming obsessed with

* It is beyond the scope of this communication to deal in detail with the mechanisms whereby the high blood lipids produce coronary disease. We would stress, however, that there are two fundamental processes which must be considered: Firstly, the process of 'atherogenesis', in which progressive narrowing of the arterial lumen is produced and secondly, the process of 'thrombogenesis', where massive thrombosis suddenly occludes the lumen of an atheromatous artery.

the need for meticulous attention to minor culinary details. Above all, he should not be allowed to become serum-cholesterol conscious' any more than the hypertensive should be allowed to become 'blood-pressure conscious'. The serum-cholesterol level varies from day to day and from week to week even more than the blood pressure. Furthermore, techniques for measuring the serum cholesterol are not easy and a reliable laboratory is necessary. Indiscriminate estimations of the serum cholesterol in all patients is undesirable but in patients with coronary disease and in the 'population at risk' the procedure is justified. But unless the reading is clearly above the mean for age and sex on at least two occasions, the patient should not be told that his serum cholesterol is too high.

3. DIETARY ADVICE TO PRIVILEGED COMMUNITIES IN GENERAL

We have mentioned the epidemiological evidence which indicates that populations consuming small amounts of fat in their diet have a low incidence of coronary heart disease. More direct evidence of the beneficial effect of fat restriction was provided in Europe during the last war.[58] During the war years, as a result of damage to farms and factories, appropriation by the militia, and the absence of skilled agricultural and food technologists, the amount of fat available for domestic consumption in Germany, Norway Sweden and Finland dropped sharply. This was accompanied by a corresponding fall in the incidence of arteriosclerotic deaths. In Denmark, as a result of failure of the export market, the local consumption of butter and eggs rose considerably and, unlike the rest of Northern Europe, no significant reduction in the incidence of arteriosclerotic deaths was noted. At the end of the war when normal fat consumption was resumed, the incidence of arteriosclerotic deaths in Germany, Norway, Sweden and Finland returned to its former level.

These observations are interesting and thought-provoking but they do not provide adequate grounds for changing national dietary customs. Other variables resulting from the state of war may not have been fully assessed. From the public point of view, it is in our opinion wrong to give general dietary advice until the indications are more certain; such advice should be deferred until adequately controlled investigations on 'populations at risk' have provided statistically significant results. At present, however, it is legitimate to draw attention to the striking increase in total fat consumption and to suggest that the higher levels are probably undesirable. The National Nutrition Council of South Africa has, in fact, made a general recommendation to this effect, and last year advised that except for very active adults 'the desirable upper limit for fat intake is probably about 30% of the total calories'[59]

Perhaps the most urgent public health project in this field is to obtain clearer evidence of the possible unfavourable effect of hydrogenation processes in the food industry. The available facts constitute a stimulus to investigation but they are not sufficiently definite to justify action in the public interest.

SUMMARY

1. It is now widely accepted that the 'Westernized' communities are experiencing an epidemic of coronary heart disease. Although genetic predisposition and sex are important and uncontrollable determining factors it is suspected that the epidemic could be controlled by modification of environmental factors.

2. The environmental factors which have been blamed include 'stress and strain', lack of physical exercise, diet, and possibly smoking. It is likely that there are others. Of the controllable factors it is postulated that none is more important or more easily modifiable than diet.

3. Many differences distinguish 'Westernized' diets from the diets of underprivileged populations, in whom the incidence of coronary heart disease is low. Some of the differences have been discussed. It is suggested that of these differences the most important may be the quantity and/or quality of fat.

4. Privileged populations consume a diet containing large quantities of fat. They have a high incidence of coronary heart disease and higher levels of blood lipid than underprivileged races. It is assumed, but not proved, that the level of blood lipid is the link between dietary fat and coronary atherosclerosis. High blood lipids may predispose to coronary heart disease through either or both of two mechanisms, namely atherogenesis and thrombogenesis.

5. The serum-cholesterol level has been chosen as the most convenient index of the blood lipid. It is, however, subject to considerable unexplained fluctuation and wrong conclusions may be drawn if too much significance is attributed to isolated readings of the serum cholesterol.

6. Evidence is reviewed which indicates that certain fats which are of vegetable and marine origin, and which are highly unsaturated, do not elevate the serum-cholesterol level and may, in fact, depress it.

7. Tentative dietary advice is offered for the management of acute cardiac infarction and for the prevention of coronary heart disease in persons particularly at risk and in privileged communities as a whole. It is emphasized that the advice is tentative and subject to modification in the light of further knowledge.

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Finally we should like to record our appreciation of the magnificent response to our appeal for volunteers by the male community of Cape Town and to the various organizations and industries, including Rotary, the City Corporation, the South African Railways and Harbours, and others, who sacrificed man-hours for the sake of these investigations.

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POLIOMYELITIS VACCINATION IN THE CAPE DIVISION 1956—57

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It is apparent from the current South African lay and medical press, and from the numerous queries addressed to the writer by both parents and medical practitioners, that there is still some confusion about the use of poliomyelitis vaccine, and hesitancy whether immunization of children should be encouraged or even permitted. This hesitancy persists notwithstanding the results of the comprehensive American Field Trials of 1954, and the confirmation provided by the subsequent safe inoculation, after a preliminary set-back, of many millions of children in the United States and elsewhere.

The profession in South Africa clearly wants to get information on the safety and effectiveness of the South African poliomyelitis vaccine. To date very few South African figures have been published, and rumours of risks and setbacks continue without convincing statistical rebuttal.

At a recent meeting of the Cape Western Branch of the Medical Association, the writer entered the discussion which followed an address on poliomyelitis vaccination by Dr. R. Turner, Senior Government Pathologist, and, after giving some preliminary figures of the recent heavy incidence of poliomyelitis in the Cape Division, attempted to correlate them with the vaccination activities in the area. As a result there has been a spontaneous demand from numerous colleagues that these figures should be published, notwithstanding the fact that they may not stand up to detailed statistical scrutiny. It seems opportune, therefore, to give a brief account of the area concerned, its population, the extent of poliomyelitis vaccination and, within the limitations of the small numbers involved, an analysis of its effect during the epidemic period which has just passed. It must, however, be stressed that many statistical defects make it impossible to draw dogmatic conclusions. The best that may possibly be achieved is a 'reasonable inference'—but after all, administrative public-health officers frequently have no option but to base their recommendations for the protection of the

public in their care on 'reasonable inference' or even 'informed guesswork'. Perhaps, too, other local-authority health departments may be encouraged to put their experiences on record so that a composite picture may be formed.

The Divisional Council of the Cape through its Combined Health Control Scheme has undertaken the responsibility for the administration of the Public Health Act throughout the whole of the Cape Division with the exception of the

TABLE I. POPULATION

European	90,000
Cape Coloured and Cape Malay	106,000
Native	22,000
Asiatic	2,000
Total	220,000

municipal areas of the City of Cape Town and Simonstown. This region embraces an area of approximately 600 square miles, includes 7 municipalities as well as the peri-urban and rural areas of the Division, and has a total estimated population of 220,000. Approximate racial grouping is shown in Table I.

POLIOMYELITIS VACCINATION

When the vaccine produced by the Poliomyelitis Research Foundation in Johannesburg became available in August 1955, the Council's Health Department had to make the decision whether to advocate the wide use of this vaccine in its area. At that time the report on the American Field Trials[1] had become available, with its great message of hope, but at the same time the public was in a state of turmoil as a result of the wide press publicity given to the preliminary set-back caused by the infection with poliomyelitis virus of some children inoculated with a portion of the first commercially produced American vaccine. There were many who advocated a 'wait-and-see' policy, on the grounds that while the incidence of poliomyelitis in South Africa had increased over the previous decade, it was still not such as to constitute a serious public-health problem. The Council's Medical Officer of Health, Dr. J. P. de Villiers, was, however, convinced that a serious outbreak was in the offing and, having satisfied himself that the vaccine was safe, resolved to advocate its widest possible use in the hope that it would also prove to be effective. The help of the press was enlisted, notices were published at schools, churches, clinics and municipal offices throughout the Division, and every effort was made to persuade parents of pre-school children to apply for the vaccine. The response from Europeans was poor—even the writer's publicized intention to inoculate his own four children at the earliest opportunity had little effect—but there was a very satisfactory reaction from the Cape Coloured community.

As a result of all these efforts, 260 European and 3,255 non-European children under 6 years of age received a first dose of vaccine during the first fortnight of September 1955. Unfortunately no vaccine became available for the second dose during that year.

A small quantity of the first batch of vaccine issued was used for a small field test of its antigenic properties. About 60 volunteers were obtained from among the European children attending the primary school of Epping Garden Village, a lower-middle-class European township. By arrangement with Dr. James Gear, Director of the Poliomyelitis Research Foundation, Johannesburg, their blood was tested for the presence of antibodies to the 3 types of poliovirus before and after a course of 2 inoculations of vaccine, with an interval of 6 weeks between them. Most of these children were found, before vaccination, to have one or more poliovirus antibody types—many had antibody against all 3 types of poliovirus. However, of those tested who lacked antibody against one or more types before vaccination, all except one developed antibody against all three types of poliovirus after vaccination.[2] (This test is being continued to determine the level of antibodies after a lapse of 18 months and to observe the effect of a third dose of vaccine.)

It was not until August 1956 that it was possible to administer a second dose to the 1955 applicants. By this time contact with the non-European vaccinated children had been lost, and only a small minority of them reported for a second dose. However, as a result of the previous year's experience, and encouraged by the preliminary results of the Epping Garden Village tests, the Council's Health Department was able to convince the majority of European parents that the vaccine was safe and antigenic, and there was no shortage of applicants. Successive batches of South

TABLE II. POLIOMYELITIS INOCULATION

	European	*Non-European*	*Total*
First dose	6,020	3,648	9,668
Second dose	5,061	954	6,015
Third dose	19	—	19

African vaccine were released, and despite the fact that there was a very steep rise in the incidence of poliomyelitis in September and subsequent months, inoculation of European children continued—first in the under-6 age-group, then in the under-10 group. By the end of April 1957 nearly 16,000 injections of South African vaccine had been given, the subcutaneous route being employed in each case—almost always over the left deltoid (Table II).

Complications of Vaccination

The majority of these inoculations were given at public sessions attended often by 200 or more children and their mothers, with complete absence of any pre-inoculation examination. Only where the child was obviously ill, or where the mother volunteered the information that the child had a temperature, was the vaccine withheld. All parents were instructed to report any untoward reaction to the Health Department without delay. In all, about a dozen parents have reported reactions, but each reported reaction has been personally investigated and has been found to have a completely adequate alternative explanation (1 case of measles, 1 of chicken-pox, several with acute follicular tonsillitis, and so on). In no case was there any reaction which could reasonably be attributed to the vaccine, nor was there any evidence of allergic reaction despite the fact that children with infantile eczema, or a history of asthma or even of previous penicillin sensitivity, were inoculated. Local reactions at the site of injection were also entirely absent, it being noted that children used, bumped, and slept on, the inoculated arm without any evidence of discomfort.

So-called 'Coincidental' Cases of Poliomyelitis

Despite the fact that the inoculation programme was continued in the face of a considerable outbreak of polio-

myelitis, only one child in the region was notified as suffering from poliomyelitis within 30 days of the date of inoculation. This child, a European male aged 9 years, received his first injection on 4 February 1957 and was notified 10 days later. At the start he had fairly severe paralysis involving both arms and the intercostal muscles, but he subsequently developed a quadriplegia; he had to be placed in a respirator. It was particularly noted that while paralysis first developed in both arms simultaneously, the right arm was somewhat more affected than the left. He had been injected subcutaneously over the left deltoid.

Although the possibility had to be considered that this child might have contracted the disease as a result of the introduction of vaccine containing live virus, it must be pointed out that the batch of vaccine had passed the rigorous safety tests as required by the revised American regulations, that as far as the writer is aware no other 'coincidental' cases were reported after the use of the same batch of vaccine, and that a number of other cases were reported in uninoculated children from the same area at about the same time. It seems reasonable to assume therefore, that this child had been exposed to infection before inoculation, and that the vaccine was not to blame. Obviously, sufficient time had not elapsed after inoculation for the child to develop any immunity to the infection.

That there was always a possibility that a child incubating the disease might be inoculated was graphically illustrated by the case of Mrs. A., a European woman aged 33, who brought her young child for inoculation to a public session on 5 September 1956, and was herself admitted to hospital with severe paralytic poliomyelitis 3 days later! Fortunately, the inoculated child remained symptom-free.

POLIOMYELITIS INCIDENCE

Table III shows the cases of poliomyelitis notified to the Cape Divisional Council during the 10-year period ending 30 June 1956. During the spring and summer of 1956/57,

TABLE III. DIVISIONAL COUNCIL OF THE CAPE: POLIOMYELITIS NOTIFICATION 1947–1956

Year ending 30 June	*European*	*Non-European*	*Total*
1947	2	—	2
1948	4	8	12
1949	4	1	5
1950	4	2	6
1951	6	5	11
1952	3	2	5
1953	9	3	12
1954	14	10	24
1955	9	24	33
1956	6	7	13

the region experienced the highest incidence of poliomyelitis in its history. In all, 200 cases, the large majority of whom were paralytic, were notified between 1 January 1956 and 31 May 1957, 181 of them occurring after 1 September 1956 (Fig. 1).

Many of these cases were severe, and there were 13 deaths (8 European, 5 Cape Coloured). In many cases the virus was isolated and typed, and it is noteworthy that every virus so typed was Type I.

Table IV shows the racial and age distribution of these 200 cases. Amongst the Europeans the incidence was not confined to young children, but extended to older children

TABLE IV. CAPE DIVISIONAL COUNCIL: POLIOMYELITIS: AGE AND RACIAL INCIDENCE, 1 JANUARY 1956 TO 31 MAY 1957

	% of total cases		*Under 6 years*		*6–12 years*		*Over 12 years*	
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
European ..	78	39	44	56·4	22	28·2	12	15·4
Cape Coloured (incl. Asiatic)	97	48½	93	95·8	2	2·1	2	2·1
Bantu	25	12½	25	100	—	—	—	—

and young adults (8 cases occurred between the age of 26 years and that of the oldest case, viz. 33). This followed the trend reported in recent years from cities of North America and Europe. On the other hand, while 61% of the total cases occurred in non-Europeans—a group in whom the disease was rarely encountered in South Africa a decade

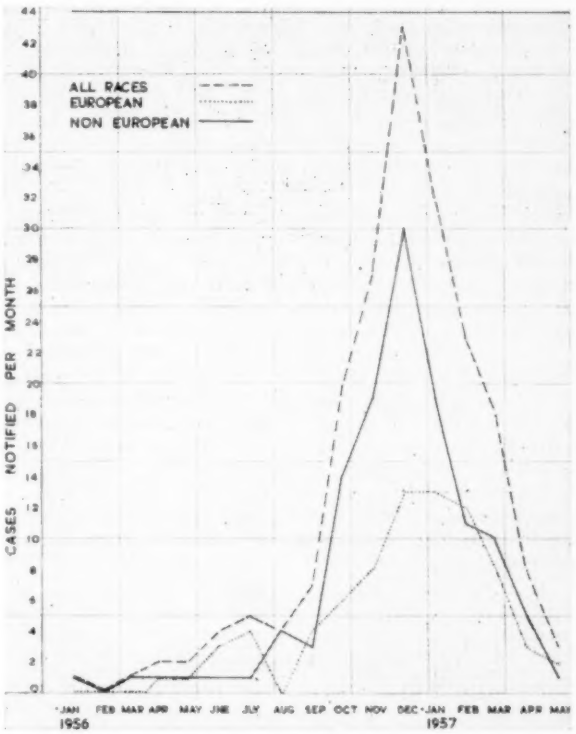
ALL RACES
EUROPEAN
NON EUROPEAN
CASES NOTIFIED PER MONTH
JAN FEB MAR APR MAY JUNE JULY AUG SEP OCT NOV DEC JAN FEB MAR APR MAY
1956
1957

Fig. 1. Cape Division. Monthly incidence of poliomyelitis notifications.

ago—practically every case in this group was a child under 6 years of age. This contrast can be readily understood when it is remembered that, while the European community has attained a reasonably high standard of hygiene, the sanitary conditions of the non-Europeans leave much room for improvement.

Effect of Vaccination on Incidence of Paralytic Poliomyelitis

Notification of each case has been followed by a careful investigation to ascertain whether the patient had previously

been vaccinated. This investigation involved not only very careful reference to the polio-vaccination records kept by the Council's Health Department, but also a detailed enquiry at the patient's home, and often from his medical attendant, as the Council has no record of the quite considerable numbers of children who have been vaccinated by private practitioners. As a result of these investigations it was ascertained that the following cases of poliomyelitis had occurred in vaccinated children:

1. European male, 12 months. First dose 28 September 1956. Notified 14 November 1956.
2. European female, 1 year 4 months. First dose 2 October 1956, second dose 8 November 1956. Notified 20 December 1956.
3. Coloured male, 1 year 7 months. First dose 14 September 1955. Notified 21 March 1956.
4. Coloured female, 3 years 6 months. First dose 15 September 1955, second dose 23 August 1956. Notified 18 December 1956.

(N.B. The 'coincidental' case referred to previously is not included here.)

All these cases had some degree of paralysis, and 2 of them were relatively severe. None died.

It should perhaps be stated that 3 cases of poliomyelitis in uninoculated children were notified from families in which one or more siblings had been given the vaccine. In each case the vaccine had been given some considerable time earlier—long enough to avoid any suspicion being cast on the vaccine. In no case did the inoculated sibling develop any symptoms.

The Council's vaccination activities have been confined to children who were not above 10 years of age in the summer poliomyelitis period just passed, and have in the main been concentrated on the European community. Accurate statistics of population are not available but, based on the estimated total European population and on the known number of European children in preparatory and primary schools in the region, a tentative estimate of the number of European children up to 10 years of age in the region places the figure at about 18,500. In this group there have been 60 cases of poliomyelitis notified, of whom 2 had been inoculated and 58 had not. (For purposes of this analysis, the 'coincidental' case referred to earlier has been regarded as uninoculated). If the estimated figure of 18,500 is accepted as the 'population at risk', and 6,000 as the number inoculated, then the incidence was as follows:

Uninoculated:	58 cases in	12,500
	464 per	100,000
Inoculated:	2 cases in	6,000
	33 per	100,000

CONCLUSIONS

It is acknowledged that the figures quoted are neither sufficiently large nor sufficiently accurate to permit of proven statistical conclusions being drawn. It is submitted, however, that it can reasonably be inferred:

1. That the South African poliomyelitis vaccine is safe, and that its use is singularly free from reactions.
2. That it is at least as effective as Salk's American vaccine has been shown to be, either during the 1954 Field Trials or during the Chicago outbreak of 1956,[3] and that a significantly high protection rate has been achieved even without the recommended third dose.
3. That, as is to be expected from the varying sanitary conditions, there are significant differences in the distribution of poliomyelitis amongst the various racial groups in the Cape Division.
4. That, in view of these differences, the decision taken to limit the offer of the vaccine to the under-6 age-group in the non-Europeans, while extending it to older European children and young adults, is supported by the epidemiological experience in the region.
5. That the possible advantages of continuing poliomyelitis vaccination, even at a period of high incidence, so far outweigh the disadvantages that a vigorous inoculation campaign should be regarded as a routine measure in dealing with an outbreak of the disease. Naturally, inter-epidemic inoculation is preferable, and should be encouraged to the limit of available vaccine supplies.

SUMMARY

A review of the poliomyelitis vaccination experience of the Health Department of the Divisional Council of the Cape is presented. The vaccination programme was continued in the face of the most severe outbreak of poliomyelitis the region has known. Figures are given showing the incidence and age-incidence of the disease in the various racial groups, and the few cases which have occurred in inoculated children are reported.

The figures quoted, admittedly inadequate, tend to indicate that the South African poliomyelitis vaccine is not only safe and free from reactions, but also at least as effective as the American Salk vaccine has been shown to be.

I wish to thank the Health Committee of the Divisional Council of the Cape, and the Council's Medical Officer of Health, Dr. J. P. de Villiers, for permission to publish this article. My thanks are also due to Dr. R. Turner, Senior Government Pathologist, and Dr. H. R. Ackermann, Medical Superintendent of the City Hospital for Infectious Diseases, Cape Town, for their encouragement and helpful criticism, and to Dr. James Gear for his invaluable share in our inoculation programme and small field test.

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REGISTRATION OF SPECIALITIES

In Government Notice No. 849 of 14 June 1957, the Minister of Health has approved of the substitution of the following new note for note (1) to rule (5) (*d*) of the rules made by the South African Medical and Dental Council under paragraph (*r*) of sub-section (2) of section 94 of the Medical, Dental and Pharmacy Act, 1928, and published in Government Notice No. 129 of 27 January 1956, as amended:

Note (1).—Experience at a hospital or institution of less than six months duration will not be regarded as satisfactory experience as prescribed in these rules, except where a person needs a lesser period for completion of the prescribed training of *thirty-six* months.

DISEASE AND POPULATION*

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In medieval times the pattern of disease was such that a physician trained in an Eastern city could practise his art in Europe, subject only to the restrictions of language and certain modifications due to climate.

The average expectation of life from birth in Europe did not exceed 19 years, and in the big cities was at times considerably less (Sand 1952). The biological balance was one of high birth-rates, high death-rates especially in infancy, diseased adults, and few old people.

The underlying causes are sufficiently well known. War, pestilence, famine, and the mass movements of people all predisposed to the great epidemics. Severe malnutrition, bad housing, and lack of cleanliness encouraged the spread of disease, and parasitic infestations, both major and minor, were universal. Above all were the effects of ignorance and superstition.

THE CHANGING PICTURE IN THE WEST

In Western Europe this pattern began to change about 250 years ago, and subsequent developments may be traced in four stages. Firstly, the slow improvement in economic and social standards extending over the 17th, 18th, and 19th centuries resulted in increased wealth, better housing, improvement in the standard of nutrition, and a general awareness of the desirability of conserving life. Secondly, from about the year 1848, came a deliberate and sustained attack on the faulty physical environment. The quantity and quality of water-supplies, the proper disposal of sewage, and improvement of housing and working conditions all received attention. Next, towards the latter part of the 19th century, may be seen increasingly successful attempts to control the biological environment, beginning with the recognition of the nature of infection and the segregation of infectious cases in 'isolation' hospitals. Intensive study of the causes of the various infectious diseases led to effective measures for their prevention.

The fourth stage—the improvement of the social environment—has reached its peak in the present century, with the organization of health and welfare services on a national scale. In this country, for example, the National Health Service, National Assistance, comprehensive education, housing and town-planning, smoke control, the protection of water sources by river boards, and the safeguarding of the quality and quantity of food have all reached full development within recent years. Above all, countries like our own can now rely on accurate statistics and stable government.

Some of the results are quite extraordinary. In England the year 1856 was a good one (Farr 1858). Potatoes were 26 lb. for 1s. in the wholesale market and beef and mutton 5½d. per lb. The population of England and Wales was 19 million, with a birth-rate of 34·5 and a death-rate of 20·5, but 20% of all deaths were due to acute fevers, and 25% of all deaths occurred in the first year of life. In that year 94,000 infants died. 'Consumption', with 49,000 deaths, accounted for 12·7% of the total mortality. Included in the principal causes of death were cholera, smallpox, typhus, and dysentery. The average expectation of life from birth of the most favoured classes was only about 45 years; but, even so, the population was rapidly increasing, in spite of emigration. It is worthy of note that 170,000 people emigrated in 1856, of whom 80,000 were Irish and 72,000 English and Welsh.

Turn now to the year of my birth, in London in 1904. According to the report of the medical officer of health it was a hot dry summer, and 'during these months there was consequently a great increase in infant deaths, due to epidemic diarrhoea'. The infant-mortality rate was 155 per 1,000 live births as compared with 24·9 today, and one-seventh of all deaths were due to infectious disease. Although the birth-rate was falling it was still high as compared with the present day, and so also was the death-rate. The expectation of life from birth was now 40-45 years for all classes. 90% of the London schoolboys examined in that year had dental caries and in 70% this was serious. Ringworm was common and the report recognized want of cleanliness and lack of food as the principal evils of children (London County Council 1904).

Today the picture is again different. The death-rate is low (11·7 per 1,000), and so also is the birth-rate at 15·0. Children and adults are healthy, and the proportion of people aged 65 and over has risen to 11%, compared with 5% in the year 1901. The principal causes of death are those of middle life, with cardiovascular disease and cancer in the lead. The death-rate from infection is insignificant, but there is increasing preoccupation with mental stress and illness, and with the neurotropic viruses.

It seems clear that we can expect relatively little further improvement in our vital statistics, unless it becomes possible to prevent or cure cancer and circulatory diseases, and indeed we must expect, in our type of society, to face an increasing burden of the elderly infirm due to the numbers now surviving to later life.

DISEASE IN UNDERDEVELOPED AREAS

We can be said to live in two worlds. Approximately one-sixth of the population of the world are now healthy, and wealthy, although perhaps not particularly wise. They are able to control their environment and, even more important, their rate of reproduction. They have adequate food, shelter and medical care.

Before proceeding further, to discuss the so-called underdeveloped areas of the world, it is necessary to stress the fallibility of the statistics. Adequate statistics are available for about one-third of the world, rough estimates obtain for another third, while the statistics for the remainder are generally inadequate (United Nations 1952). It should be noted, also, that much of the data on which reliance is placed today was collected in the years immediately after the war. It is possible, therefore, that this reflects the fluctuations following the second world war and does not truly mirror the conditions which would prevail in times of stability.

The population of the world in 1954, according to the United Nations Statistical Office, was 2,652 million (United Nations 1955). More than half (1,451 million) lived in Asia, the remainder being distributed as follows:

Europe	404	million
America	357	,,
Africa	210	,,
U.S.S.R. (not included in Europe or Asia)	214	,,
Oceania	14	,,

In parts of Asia and Africa the disease picture resembles that of medieval Europe, for the individual may suffer from five or six diseases. The people are at risk from the killing diseases of temperate climates in addition to 'tropical' diseases. Severe malnutrition is common, and added to this are the harsh physical environment, poverty and ignorance, bad housing, and often the crumbling of the ancient social patterns. Birth-rates are high, but so are death-rates, and the natural picture of population has been one of slow increase with sharp cuts due to famines or epidemics.

That was the picture in many areas until about ten years ago. Indeed, the year 1947 may be considered, like the year of the Black Death, a turning-point for the world, for it was then that the first effects of the mass attack on malaria became apparent.

It is now possible to launch attacks on disease at national and international levels, and on several fronts at the same time. Environmental sanitation is rapidly being improved, and especially the chlorination of water-supplies, so that cholera, dysentery, and enteric fever, are declining in importance. Malaria, predominantly a disease of rural areas, with 300 million cases a year in the world and 3 million deaths, is now being attacked through the mosquito at a cost of 15-50 cents per head. In Ceylon in one year, 1947, the death-rate was almost halved from this cause alone. Yaws, which affects most land areas between the Tropics, can be cured by one injection of penicillin at a cost of 2 dollars per head. Tuberculosis, the greatest killer of all, is now being assailed in many ways, including immunization with B.C.G.

* Abridged from an address given to the Science Masters' Association on 3 January 1957. Reprinted from *The Lancet* (1957): **1**, 749.

Bilharzia, estimated in 1947 to affect 114 million people (Stoll 1947), is receiving increasing attention, and so also is hookworm, which by reason of its distribution from 36° north to 30° south of the Equator can affect half of the inhabitants of the world. Trachoma, a serious cause of blindness in both arid and wet tropical and sub-tropical regions, is readily amenable to modern therapeutic and preventive measures.

Malnutrition, as evidenced by beriberi, pellagra, scurvy, rickets, and nutritional anaemia, in addition to protein deficiencies under various titles such as that of kwashiorkor, is now being treated on a vast scale by the provision of vitamins and dried milk.

The results are, at first sight, catastrophic, for, while death-rates are being cut by half, birth-rates remain high or are rising. Examples of the high birth-rates prevailing in various parts of the world are the following:

Venezuela	in 1955	..	..	47·1
Mexico	in 1954	..	..	46·4
Ceylon	in 1955	..	..	37·9
Chile	in 1955	..	..	35·0
India	in 1955	..	..	30·5

The corresponding rate for England and Wales is 15 per 1,000 population.

Many more people are now surviving to adult life and reproductive ages as a direct result of the control of the killing diseases. But other factors are at work also. The control of famine, the reduction of losses in small and local wars, improved transport and communications, rising economic standards, education, and good government, are all playing a part.

PROBLEMS OF POPULATION

In Puerto Rico the expectation of life at birth has increased by over one year per annum since 1940. This is at least twice, and often three times, as high as the maximum national rates ever recorded in the populations of the West. Even so, the Puerto Rico rates were doubled by Japan between the years 1947 and 1950 and trebled by Ceylon between the years 1946 and 1949 (Stolnitz 1955).

In India the infant-mortality rate in 1910 was 260 per 1,000 live births. In 1951 it had fallen to 116. If this rate can be reduced to 35 within the next twenty years, as seems possible, it will have fallen from 260 to 35 in seventy years.

In Brazil the crude death-rate for 1940-50 was between 19 and 20 per 1,000, whereas the birth-rate for the same period was between 43 and 44. The rate of natural increase was 23 per 1,000, and the expectation of life from birth has risen to 42 or 43 years.

In the Sudan the pilot census of 1953 showed a crude death-rate of 37 per 1,000 and a birth-rate of 56 per 1,000.

Many parts of the world are, therefore, able to demonstrate changes in their vital statistics over a period of ten to fifteen years which took us seventy or more years to accomplish. They can, in fact, go on and beat the Western figures for low death-rates because of their high proportion of young people.

It is at first sight odd that Oceania should have the highest rate of increase at 2·5% per annum, but this is explained by immigration. The United States of America and China come next with an increase of 2% per annum, followed by Asia, Africa, and the U.S.S.R. with about 1·5% per annum. Europe is well behind with 0·7% per annum.

The average world population increase is about 1·5% per annum, and when it was thought that food production was only increasing at the rate of 1% per annum (0·04% in the Far East), the picture gave cause for immediate alarm. The latest reports of food production, both as to quantity and stability of output, are more reassuring (Food and Agriculture Organisation 1956). Even so, after allowing for all statistical fallacies, 100,000 births a day are something to be reckoned with.

The most recent World Health Organisation statistics show a tendency to stabilization of birth and death rates in the period 1951-55, with a slight overall decrease (World Health Organisation 1956). The infant-mortality rate is declining rapidly, and in 1955 Sweden set up a new record with 17 infant deaths per 1,000 liveborn. This is the lowest rate ever recorded in the world, and can be contrasted with 422 per 1,000 in Maceio in Brazil in 1943 and 353 in Saigon in Vietnam in 1947. As the improvement of infant-mortality rates turns on such matters as family income, feeding, housing, sanitation, the working conditions of women, and the control of mass diseases including malaria, it is a reliable indication of the measure of improvement of those conditions also.

It should be remembered that only certain countries have an immediate population problem. Egypt, for example, is heavily overpopulated, whereas Iraq and Persia are not. In general, the problem appears in its most acute form in islands. Quality, as well as quantity, is involved. The fertility of the people of agricultural areas is particularly affected, and that of market-gardeners and paddy and coconut workers in Ceylon is now 40% higher than that of the people of Ceylon as a whole. That is, of course, primarily due to the control of malaria.

When discussing the growth of population it is tempting to blame the doctors for upsetting the balance of Nature, and it is often forgotten that the people are themselves demanding the prevention of disease. There are many other factors involved, besides the advance of medical science. There is, for instance, the cultural pattern which demands large families; the duty, often reinforced by religion, to perpetuate the family; and the need of agricultural peoples, using primitive methods, for large families if they are to survive. The removal of taboos on sexual intercourse, which in some parts of India covered as many as 120 days in a year (Chandrasekaran 1954), must also be taken into consideration. It is possible also that the biologists may, as a result of their studies of population changes in animals, have something to contribute to our knowledge of the natural rhythm of growth of human populations.

POPULATION CONTROL

The most obvious remedy is some form of birth-control which would be widely acceptable. The various methods include total abstention from sexual intercourse; partial abstention by the rhythm method, which requires an ability to count, the wish to cooperate, and some elementary knowledge of biology; mechanical methods which require a trained physician working in maternity and child-welfare centres (which do not yet exist in many areas); and the 'little pill,' containing some substance which will interfere with the maturation of the sex cells or embryo. If, and when, the latter becomes available it will need to be tested, manufactured on a commercial scale and sold at prices which the people can afford—its use being acceptable to them.

We must face the fact that there is no short cut, and that the present rapid growths of population will involve a period of turbulence and instability for the areas concerned for perhaps fifty years or more.

Meanwhile the results of the medical advances have not all been bad. Malaria control has resulted in large tracts of land being brought into cultivation, and the improved health of the workers has meant increased production, both of food and of industrial products. The control of hookworm alone is said to increase the working efficiency of affected people by 25% or more, and similar results will attend freedom from other diseases. The people most concerned are those living in the rural areas of the world and responsible for the production of food. Improvements in transport and communications mean, in turn, the possibility of improved distribution of food.

There is also the changing attitude towards women, with outlets other than marriage, in the fields of teaching, nursing, and industry. Nor must it be forgotten that the means to combat ignorance and prejudice are becoming increasingly available, both by the extension of formal education and by the radio and television.

CONCLUSION

The first and most urgent need is to obtain true and reliable statistics of population and of food production, so that we may have an accurate world picture. There is also an urgent need for combined action to study the problems of areas where the pressures seem to be greatest. The persons required for such investigations include geographers, biologists, agriculturists, economists, veterinarians, anthropologists, administrators, and medical men.

Until we have full knowledge of the problems of population growth it will be as well to observe the ancient maxim, 'festina lente,' for the happiness of a people does not necessarily turn only on an increased expectation of life.

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HISTORICAL SURVEY OF MEDICAL RESEARCH IN SOUTH AFRICA

The Report* of the Commission of Enquiry into the Coordination of Medical Research contains the following description of the 'beginnings of medical and veterinary research in South Africa'. Medical research received a tremendous impetus in the closing decades of the 19th century, when the work of European scientists such as Robert Koch and Louis Pasteur was establishing a genuine scientific aetiological basis for disease and uncovering vast new worlds. At first research developed entirely around individuals: in 1878 the *British Medical Journal* observed that Government research moneys were paid entirely to specialists such as Sir Oliver Lodge and E. A. Schäfer; but the 1880s saw committees formed for 'collective investigations' and in 1887 the Royal College of Physicians of Edinburgh appointed a committee to consider the formation of a medical research laboratory. In South Africa organized medical research owed its inception to the ravages of stock diseases such as rinderpest, horse-sickness, red-water, lungsickness, nagana, etc. rather than to those of the great human epidemics of the 18th and 19th centuries at the Cape.

Individual research, and indeed a curiosity to investigate scientific phenomena, had been a feature of the Cape medical fraternity since the heyday of the Old Somerset Hospital. The first epidemics of leprosy and smallpox and the professional associations at the Old Somerset Hospital and the meetings of the South African Medical Society in the 1830s stimulated the medical practitioners of the Cape to enquire more deeply into the problems of everyday practice. Regular meteorological observations were made—the weather was still implicated as a possible aetiological factor in disease—and papers of a philosophical and inquiring nature were read. Practitioners were invited to witness 'Capital' operations at the hospital, and registers of the diseases found in the course of practice were kept as 'the first steps towards reducing the medical statistics of the Colony to a science, for without it all must be conjecture.'†

Despite this, the references to research remained pleas in favour of it rather than reports of the work undertaken. During the 1840 smallpox epidemic, the Government even went so far as to offer a handsome prize for anyone who produced a variola vaccine from the cow itself, since a high rate of failure was being recorded with the material obtained from Europe. Detailed advice on the techniques employed by Jenner and other European workers was gazetted in this connection, but there appears to have been no response to the venture. Several years later, the first editorial of the *Cape Town Medical Gazette* urged investigation of the Cape's diseases—'what of Consumption, Rheumatism and Leprosy, a disease very prevalent in this Colony'—and of the Colony's unique materia medica. In this latter field, however, much original research was undertaken by the Drs. Leisching, father (1757-1841) and son (1802-1843), and especially by Carl Wilhelm Ludwig Pappe, M.D. (1802-1862), first professor of Botany in the South African College, who collected over 100 species of indigenous plants used for their medical qualities at the Cape and published a scientific dissertation on them in 1850 In leprosy also, original research was conducted by workers such as Samuel Patton Impey, M.D. (1856-1928), the Colonial-born superintendent of the Robben Island Leper Asylum, who won the Sydenham Prize for an essay on leprosy in the 1890s. The Leprosy Commission appointed by the Cape Parliament in 1893 inquired exhaustively into the disease, its aetiology no less than its public health implications, and the report, presented to Parliament in 1895, embodies some of the first conclusions reached as a result of research into a specific disease in South Africa.

* U.G. 14/1957. Government Printer, Pretoria.
† Cape Almanac for 1832, page 115.

EDINGTON

By that date the foundations of organized research had been laid, for Dr. Alexander Edington had already commenced work at the Colonial Bacteriological Institute in Grahamstown in 1891. This laboratory was established 'in order to investigate some of the more important diseases affecting stock, which have of late years increased so seriously that stock farming has become a most precarious pursuit.'* Whilst the Institute's primary duty therefore lay in the veterinary field, it assumed medical functions as well, and soon became the first public health laboratory in South Africa. The decision to place the Institute in the Eastern Province brought to a successful conclusion the long struggle of Dr. William Guybon Atherstone (1814-1898), the veteran Grahamstown practitioner and probably the first man outside of America and Europe to use ether as a general anaesthetic, to have a laboratory erected in Grahamstown. The unsuitability of the site was soon realized, when the lack of gas in Grahamstown made laboratory procedures difficult, and the city's relative inaccessibility severely hampered the field work of its Director elsewhere in the Colony.†

Edington must be regarded as one of South Africa's first medical 'specialists' as well as one of the first full-time public-health officers of the Cape Colony. He was also the first editor of the *South African Medical Association's Journal*. As Director of the Colonial Bacteriological Institute in Grahamstown for 13 years (1891-1904) he carried out valuable research on medical and veterinary problems. He also travelled extensively throughout South Africa and even visited Mauritius in 1902 to investigate an outbreak of trypanosomiasis in cattle.

The Colonial Bacteriological Institute was housed in a building that had been previously the depot of the Royal Engineers in Grahamstown. The building had once been used as an agricultural college, and after several alterations had been carried out there was ample space for the laboratory, its library and other departments. Edington conducted research into most of the common stock diseases, but achieved notable success by his investigations into horse-sickness. He collaborated with Robert Koch during the latter's visits to South Africa in 1896 and to Rhodesia in 1903.

Veterinary work could no more be divorced entirely from purely medical work than routine laboratory procedures from research, and within a year of Dr. Edington's arrival at Grahamstown the Colonial Government requested him to arrange for the manufacture and storage of calf-lymph for vaccination purposes, since smallpox had again broken out.‡ By July 1893 production was in full swing, in time to supply the needs of the Transvaal for the compulsory vaccination programme instituted after the severe epidemic in Johannesburg. Edington reported that 99% of the vaccinations performed were successful. All the South African states were drawing on the Institute for supplies; 'as a Vaccine Institution alone, the Institute has justified its existence.' In the same report, Dr. Edington reported the first properly identified cases of actinomycosis ('woody tongue') and bovine tuberculosis in South Africa.

* Parliamentary Papers (Cape): Report of the Colonial Bacteriological Institute for 1892 (G24F—1893).
† *Idem:* Report of the Director of the Colonial Bacteriological Institute for 1896 (G30—1897).
‡ Cape Parliamentary Papers (Cape): Report of the Colonial Bacteriologist for 1893 (G5—1894).

When a full-time health officer was appointed to the Cape Colony in 1895, Dr. Edington initiated a routine pathological laboratory service 'on lines similar to those obtaining in England, France and America, and it has been found that the medical practitioners of South Africa and the Medical Officer of the Colony take advantage of the scientific opportunity afforded by this scheme for the diagnosis of doubtful cases.'* Soon this service was extended to general examinations of exudations and tissues and public-health investigations such as testing the purity of water and milk. This expansion was made possible by the appointment of a medical assistant, Dr. Robert Sinclair Black, and a veterinarian, Mr. Pye, M.R.C.V.S., to the staff. At the Government's request, routine preparation of the newly-discovered antitoxin for diphtheria was commenced, and in 1896 a diagnostic service was established for this disease as well.† By 1897 the Colonial Bacteriological Institute was firmly established as the pathological laboratory and vaccine station of the Cape Colony. It remained as such until Dr. Edington's retirement from the service in 1904, and became thereafter an institute devoted entirely to veterinary work.

A similar institution was commenced in Pietermaritzburg by the Natal Government in 1897. This was the Allerton Veterinary Laboratory at Town Bush, where facilities for bacteriological research were provided. The stimulus for this laboratory probably came from the stationing at Fort Napier of Surgeon-Maior (later Sir) David Bruce,‡ who investigated the serious outbreaks of nagana in Zululand for the Government between 1894 and 1897. Here he was able to demonstrate for the first time that the tsetse fly carried the trypanosome (thereafter called *Trypanosoma brucei*), which caused nagana. After the South African War Dr. W. Watkins-Pitchford was stationed here as Assistant Bacteriologist to the Natal Government, and 'through his excellent services the laboratory became recognized as the official public health, medico-legal and pathological institute for Natal.'§

WATKINS-PITCHFORD

Watkins-Pitchford first came to South Africa in 1900 as bacteriologist to the No. 7 General Military Hospital with the British forces. His association with Natal after the conclusion of the Anglo-Boer War probably arose from the fact that his brother, Colonel H. Watkins-Pitchford, was the Natal Veterinary Bacteriologist, whose headquarters were also at the Allerton Veterinary Laboratory—thus again ensuring a close integration of medical and veterinary research.

The post to which Watkins-Pitchford was appointed at Pietermaritzburg after the war was later modified, and after 1907 he was styled Government Pathologist and Analyst. In 1911 he was transferred to Johannesburg as Government Pathologist and Bacteriologist there, and in the following year he was named as Director of the newly-erected research laboratories for South Africa—The South African Institute for Medical Research,‖ a post he held until his retirement through ill-health in 1926. On the Witwatersrand he became Honorary Pathologist to the Johannesburg General Hospital, and the first Chairman of the Miner's Phthisis Medical Bureau when it was established in 1916.

THEILER

In the Transvaal, as in the Cape and in Natal, the first serious attempts to undertake medical (and veterinary) research date from the early part of the last decade of the 19th century. Among the early pioneers Dr. Theiler (later Sir Arnold Theiler) played a leading part. He had arrived from Switzerland in 1891 as a qualified veterinary surgeon, with no specialized training but with unbounded energy, ambition and enthusiasm for research. His first opportunity came when a serious outbreak of smallpox occurred in Johannesburg. Theiler's name was brought to the notice of the authorities as that of a man with experience in the production of smallpox vaccine. He was engaged by the Smallpox Committee and within a month was supplying sufficient vaccine for local requirements. Within 6 months the outbreak had been brought under control.

Early in 1896 rinderpest invaded the Transvaal from the north. Theiler was summoned by President Kruger and sent to Rhodesia to investigate. And so started his research career which was to continue without interruption until the time of his death in 1936.

In 1897 a small laboratory was built for him at Daspoort near Pretoria. Here he studied not only rinderpest but most of the other animal diseases which occurred in the country, such as horse-sickness, glanders, pleuro-pneumonia, red-water, black-quarter and many others. The laboratory at Daspoort soon became too small for its purpose and the Responsible Government of the Transvaal in 1907 decided to erect a much more spacious institute. On 1 October 1908, the buildings at Onderstepoort were opened.

MILESTONES

In this brief sketch of the early evolution of medical and veterinary research in South Africa we have observed four milestones which stand out as monuments to the early pioneers in this field. These four milestones are: (1) The Colonial Bacteriological Institute in Grahamstown, erected in 1891; (2) the Veterinary Laboratory at Allerton near Pietermaritzburg, erected in 1897; (3) the Onderstepoort Veterinary Laboratory near Pretoria, erected in 1908; and (4) the South African Institute for Medical Research in Johannesburg, erected in 1912. The latter two institutes still play the leading role in the respective fields of veterinary and medical research in South Africa and both have acquired world-wide fame.

ESTABLISHMENT OF THE DEPARTMENT OF HEALTH

Prior to the formation of the Union, each of the four colonies had its own health legislation and administration. The South Africa Act, of 1909, in terms of which the Union was established, assigned to each of the four provincial administrations certain functions, including the administration of hospitals and charitable institutions. Health services, however, were not mentioned in the Act, but the control of infectious diseases seems to have been regarded as a Union Government function. As there was no Union-wide legislation, however, such laws as were in existence in each of the four former colonies remained in operation in the respective provinces, thus resulting in lack of uniformity, apparently leading to some confusion.

The position was obviously not very satisfactory and the then Minister of the Interior, Sir Thomas Watt, accordingly called a conference of the Administrators of the various provinces and other interested parties. This conference met under his chairmanship in Bloemfontein in September 1918. It was unanimously decided that it was necessary to establish a separate Ministry of Health and a department having suitable power to administer health matters under legislation which would be applicable throughout the Union.

Immediately following this the world-wide influenza epidemic occurred and took heavy toll in South Africa as in many other countries. The establishment of the Union Health Department in 1919 has often been attributed to this epidemic. In actual fact it would seem that, although the epidemic did not precede the conference at which it was decided to establish the Department, it did indirectly have the effect of considerably expediting the passage of the Bill through Parliament during the session of 1919. Sections 2 and 5 of the Public Health Act (No. 36 of 1919), providing for the establishment of the Department under the control of a Minister of Health and the appointment and duties of the first Chief Health Officer, became operative on 1 July 1919, while the other provisions of the Act came into operation on 1 January 1920. The first Chief Health Officer was Dr. J. Alexander Mitchell, whose appointment was gazetted on 4 July 1919.

From these small beginnings and from the limited functions which it then exercised the Department has expanded rapidly and taken on wider functions and greater reponsibilities until today it is one of the largest State departments—with a staff of some 9,000 persons of various ranks, apart from nearly 400 part-time district surgeons, some 25,000 beds in its various hospitals, and an annual budget of nearly £10,000,000.

The Commission sets out section 3 of the Public Health Act 1928 (as amended), which enumerates certain functions of the Department

* *Idem:* Report of the Director of the Colonial Bacteriological Institute for 1895 (G9—1896.)

† *Idem:* Report of the Director of the Colonial Bacteriological Institute for 1896 (G30—1897).

‡ *South African Medical Directory* for 1897, page 125.

§ Curson, H. H. (1933): S. Afr. Med. J., **7**, 745.

‖ Transvaal Med. J. (1912): **8**, 91.

of Health and lays stress on the words, 'To promote or carry out researches and investigations in connection with the prevention or treatment of human diseases'.

ESTABLISHMENT OF THE MEDICAL AND DENTAL SCHOOLS IN SOUTH AFRICA

At the beginning of the century there were no medical or dental schools in South Africa, but intending doctors could as early as 1905 complete their first year's course at the South African College (now the University of Cape Town). The remaining parts of the medical curriculum had to be completed at an overseas medical school. Largely through the efforts of eminent and influential medical practitioners in Cape Town and Johannesburg, and with the cooperation of the teaching institutions out of which the Universities of Cape Town and the Witwatersrand grew, medical schools were established in these two centres. These two schools, after initial struggles in temporary accommodation and with woefully deficient facilities according to present-day standards, rapidly expanded. In Cape Town the first graduates received their degrees in 1922, followed 2 years later by the first graduates in Johannesburg.

The first dental school in South Africa was established in Johannesburg. Plans for such a school first formulated by the University of the Witwatersrand in 1923 bore fruit after the provison of hospital facilities in the Memorial Hospital built by the Joint Council of the Red Cross Society and the Order of the Hospital of St. John of Jerusalem. The increasing demands on the hospital and the growing number of students soon made it necessary to provide more adequate hospital facilities. This was done by the erection of a new Dental Hospital, the building of which was completed in 1942. The site on which this hospital was built was, however, later required by the Railways Administration. The building was therefore expropriated and the present Dental Hospital built in the University grounds at Milner Park. This hospital completed in 1952 affords excellent modern clinical and laboratory facilities for education and research on dental diseases. Within the University of the Witwatersrand dentistry was administratively separated from medicine by the creation, in 1930, of the Faculty of Dentistry.

The third medical school established in South Africa was that at Pretoria. Its establishment was approved in principle by the Pretoria University Council in 1937, but it was not until 1943 that the Faculty of Medicine came into being, and the first graduates in medicine at Pretoria were capped in 1947.

The University of Pretoria is the only other in South Africa, apart from the University of the Witwatersrand, with a dental faculty. The creation of this faculty was agreed to in 1949 and the first dental students were accepted in 1950. Initially there was no dental hospital, the students gaining their clinical experience at the Pretoria Dental Clinic, but since 1951 the University's dental hospital situated in the grounds of the general hospital has been available.

As long ago as 1922 efforts were made to start a private school to train non-European doctors in Durban, but the effort had to be abandoned. Many years were to elapse before the establishment of a medical school for these students came about. Once again the efforts of local practitioners together with those of the University finally succeeded and in 1951 the first medical students were enrolled at Durban. The main building for this school which is 'primarily for non-Europeans' was officially opened in 1955, but the clinical facilities in the adjacent teaching hospital (King Edward VIII) have not yet been fully developed.

The Commission's Report then referred to the recent establishment of the youngest medical faculty—that of the University of Stellenbosch—and of the Carl Bremer Hospital, Bellville, Cape, which is being used as the University hospital pending the erection of a permanent teaching hospital elsewhere in the northern municipal area of Cape Town.

The advent of free hospitalization brought with it a change-over from an honorary system of staffing to a salaried one. Although minor differences in detail exist in the agreements between the various Universities and Provincial Administrations the principle of the joint staff agreements are similar. The Universities and the Provincial Administrations are joint employers of the staff which is employed on a whole-time or part-time (sessional) basis. The Universities' share in the expenditure on salaries is for teaching and research, whilst the Provincial Administrations pay for the care of the sick. In the case of the Durban Medical School the details of staffing in the clinical departments have not yet been completed. The change in staffing arrangements referred to above came about between 1943 and 1952. This period, too, brought an increase in support for research through the Council for Scientific and Industrial Research (C.S.I.R.). These two factors have contributed greatly to the research output at all the medical schools.

It is only in recent years that facilities have been developed for the training of post-graduates in the medical schools. Higher degrees and diplomas in many specialties are now being offered at the three older schools. There is no doubt that Durban and Stellenbosch, too, will soon develop their post-graduate facilities.

The growth of the South African Medical and Dental Schools can be gauged from the numbers of students graduating from these schools in 1955:

M.B., Ch.B.	247
Higher degrees including M.D., Masterships, and diplomas in special branches	39

ESTABLISHMENT OF THE COUNCIL FOR SCIENTIFIC AND INDUSTRIAL RESEARCH

The South African Council for Scientific and Industrial Research (C.S.I.R.) was established by Act of Parliament in 1945 (No. 33 of 1945). It replaced the previous Government bodies which had been set up to support research in South Africa. Special mention should be made of the Research Grants Board, which had been instituted in 1918 and which, in 1938, was superseded by a new organization, consisting of two bodies, the National Research Council and the National Research Board. The Council was purely an advisory body, while the Board's duties were administrative in character and included those of the former Research Grants Board.

The functions of the abovementioned organizations were to advise the Government regarding the encouragement of research in the Union and to administer all government grants in aid of research, as well as any other funds placed at its disposal for a similar purpose. However, the support which these organizations received from the Government was so meagre that very little could be done for the advancement of science. Despite this, however, it should be noted that these bodies also made small grants for medical research.

The War of 1939-45 brought overwhelming proof of the need for and the value of scientific research, and in most countries steps were taken to place research on a proper footing and to make adequate provision for its support. In South Africa Dr. B. F. J. Schonland was invited by the Prime Minister to submit a scheme for a national research organization. His proposals were accepted by the Government; the necessary legislation was passed by Parliament and Dr. Schonland was appointed as the first President of the Council for Scientific and Industrial Research.

The C.S.I.R. includes in its sphere of interest academic or fundamental research, applied research including industrial research, and medical research. It excludes agricultural research, which falls under the Department of Agriculture, and social research, which is sponsored by the Department of Education, Arts and Science.

The establishment of the C.S.I.R. has meant that for the first time in the history of medical science in South Africa more adequate funds have become available for research. For our present purpose it is sufficient to point out that support for medical research is given by the C.S.I.R. in the following ways: By the establishment and maintenance of Medical Research Units at the Universities and at other institutions; by awards to research workers for (*a*) assistants, (*b*) running expenses, (*c*) capital expenditure, (*d*) publications, and by an annual grant to the South African Institute for Medical Research.

In this brief review of the early history and the development of medical research, in the wider sense of the term, in South Africa we have seen that the main activities have become centered at (1) Onderstepoort (for veterinary research), (2) the South African Institute for Medical Research, and (3) the Council for Scientific and Industrial Research.

In addition research is being carried out under the aegis of the Department of Health and some at the Medical Schools independently of any assistance from the C.S.I.R. The Chamber of Mines and, to a small extent, one or two Government Departments are also involved in certain aspects of medical research.

THE DOCTOR'S HOBBIES: AN APPEAL

MORRIS J. COHEN, M.D., M.R.C.P.

Convener, Arts, Crafts & Hobbies Section, South African Medical Congress, Durban, 1957

In the introduction to the life of the famous artist-craftsman, Carl Faberge, his daughter Eugenie remarks: 'We live at such a feverish pace in our modern world that we sometimes forget that not so long ago craftsmen were still able to work unhurriedly and with real love of creation. It is important that we should be reminded that we are in danger of losing a heritage of incalculable value. As more is done for man, so he seems more and more incapable of doing anything for himself.'

Doctors are notoriously 'short of time'. We are on our toes most days of the week and, in more instances than not, the nights as well. Perhaps in no other profession or calling is the responsibility greater. Long hours, irregular meals and short periods of oft-disturbed sleep, states of tension and great distances to travel, heavy professional bags to carry in liftless buildings, create a tiredness and a fertile field for the development of coronary disease. In this respect, we are probably worse off than high-pressure business executives.

So away with the tendency to thrombosis, ulcers and premature geriatric conditions! Set to, and learn to do something with your hands and so occupy your mind in spheres different from your daily work. 'No time', you say, 'Make time,' say I.

And if you feel that you are not apt at making something, then you can interest yourself in collecting one or several of ten thousand articles. Some may even turn out to be profitable. But that need not to be the motive. Or you may consider it worth while to feed and breed tropical fish or birds, cultivate aloes, carnations or tulips, or collect and study orchids. And, as the King said in the play, 'Etcetera, etcetera, etcetera.' Whatever your interests, you will make friends, both professional and lay, with others who are interested in similar pursuits.

Now this is an appeal. Durban at its Medical Congress in September of this year will welcome all who wish to exhibit their hobbies, creations, or collections. This invitation is extended not only to doctors, practising or retired, but also to their families.

The rest of the profession and the public particularly will be keen to see what you do in your spare moments—time wrested from your daily routine. Our distinguished overseas visitors will be more than interested; the wife of one of them, herself an artist, has agreed to perform the opening ceremony of the Hobbies Section.

Proudly we claim that we are one of the very few countries that features such a section at its Medical Congresses.

To those who have not yet exhibited at our Congresses because of shyness or a fear that what they might offer is not good enough, my advice is to fear no criticism. We are all amateurs, doing what we do for the love of it. Your craftmanship or your collection may show merit and may command admiration and respect. Show us your wife's work in knitting, weaving, embroidery, lace work, tapestry, pewter work, painting, carving or leather. Or even the fancy waistcoat she made for you. Your child's efforts at puppetry, doll making, painting, modelling or construction of a miniature stage will thrill us. We set no limit to the type of work or collections we want to display.

Durban was the venue of the first Hobbies Section at Congress in 1946. We should like the 1957 show to be fully representative and a bumper one. The exhibition will be held in the Art Gallery, City Hall, so central, so well lit and so admirably suited for such exhibitions. It will be open to the public all day during the whole of the Congress period. This is an innovation.

A comprehensive catalogue will be published. Exhibitors are strongly advised to return the necessary information in the circular which has been sent out for the purpose, and as soon as possible. The ceiling day for inclusion in the catalogue will be 31 July 1957.

We should like to feature the writings of doctors on non-medical subjects. Books, pamphlets, magazine and newspaper articles will be accepted. Plays, poems, biographies, children's stories, anecdotes, will be welcome. We wish to show how cosmopolitan your interests are. And if your work has not yet been published, we shall be glad to exhibit your manuscripts. Needless to say, subjects in both official languages will be acceptable; or if written in classical Latin or Greek; or for that matter in any language.

If you make furniture or toys, bind books, frame or paint pictures, do calligraphy, carve in any material, do sculpture in any medium, take photographs, work in stone, brass, pewter, copper, glass, shells, wrought-iron or leather; design materials, make etchings or woodcuts, work your own printing press, create jewels or ornaments of any kind from any material, we shall be glad to have them. If you make models of any kind, boats and ships, motor cars, aeroplanes, submarines, windmills, engines, pumps or grain elevators, send them along. Then there is pottery and ceramics.

And if you are a collector, let us see your gorgeous cameos, netsukes, medallions, coins and stamps. The humble lithops may create as much interest as the rare orchid or dwarf tree. The flotsam and jetsam you picked up as a beachcomber and turned into a shapely lamp or ash tray, will show us your ingenuity. Perhaps you possess a necklace of old Arab beads gleaned from the beaches of the Wild Coast. Your collection of old Cape silver will add interest. Perhaps you are a cartoonist. If so, give us all the pleasure of a quiet chuckle.

If you are good at gadgets, medical or other, you should show them.

Items of medical interest concerning medicines of the Bushman would create interest. So would a witch-doctor's complete outfit. Antique medical instruments and accessories, such as bleeding cups would attract attention.

This does not exhaust the list of subjects you may be making or collecting. These are some that came to my mind.

There may come a time for you to retire—willingly or unwillingly. Some may well ask, retire to what? Days filled with boredom, if you are not a hobbyist. Golf alone may not be enough, nor bowls, nor fishing nor travel. The real answer is a hobby. Indeed, it may be a good excuse to travel to foreign exhibitions, to make new contacts, to gain new views and part with yours, to exchange or collect materials. Your hobby may first gradually, and later totally, supplant your professional interests.

And finally, you will not only derive pleasure from your hobby, and a measure of relaxation as well, but your enthusiasm may infect others, and your exhibits give great joy to many thousands.

SOUTH AFRICAN MEDICAL CONGRESS : SUID-AFRIKAANSE MEDIESE KONGRES, DURBAN 16—21 SEPT. 1957

THE BANQUET

On Tuesday evening 17 September at 8 p.m. a Banquet for medical graduates will be held in the City Hall, Durban, at which his Excellency the Governor-General has been pleased to signify his intention to be present.

This function will give members of the Association an opportunity of hearing and informally meeting our distinguished overseas visitors.

DIE FEESMAALTYD

'n Feesmaaltyd vir Mediese Gegradueerdes sal op Dinsdagaand 17 September in die Stadsaal, Durban, gehou word by welke geleentheid Sy Eksellensie, die Goewerneur-Generaal, sy voorneme uitgespreek het om aanwesig te wees. Hierdie funksie sal lede van die Vereniging 'n geleentheid gee om ons gesiene oorsese gaste informeel aan te hoor en te ontmoet. Spesiale reëlings sal getref word om te verseker dat mede-alumni, lede van Groepe en vriende

Special arrangement will be made to ensure that fellow alumni, members of Groups, and friends may sit together and have an opportunity of renewing acquaintanceship.

The seating arrangements only permit 600 (six hundred) members to attend.

THE BALL

The Congress Ball will be held on Friday evening 20 September at 8.30 p.m. in the City Hall, Durban. Special arrangements have been made to ensure that this function will be the unique highlight of the social programme of the 1957 Medical Congress.

The accommodation will be limited to 500 couples.

IMPORTANT ANNOUNCEMENT

Owing to the number of requests for tickets for both the Banquet and the Ball, accommodation is now limited. Tickets for both these functions will be issued strictly in the order in which intention cards are received.

Members attending Congress are therefore asked to make their applications as soon as possible to prevent disappointment.

CONGRESS FEES

In order to avoid congestion and to expedite registration on the opening morning, members are requested to send the registration fee of £3 3s. (three guineas), the amount for the Banquet tickets, £2 2s. (two guineas), which includes all wines and spirits before and during dinner, and the amount of the Ball tickets, £3 3s. (three guineas) for a double ticket, if possible before the date of Congress, to the Hon. Treasurer, 53 Medical Centre, Field Street, Durban.

B. Crowhurst Archer
S. Disler
Honorary Organizing Secretaries,
41*st South African Medical Congress*

53 Medical Centre
Field Street
Durban

bymekaar kan sit en 'n geleentheid sal hê om vriendskapsbande te hernieu.

Daar is slegs sitplek vir 600 (seshonderd) lede.

DIE DANSPARTY

Die Kongresdansparty sal op Vrydagaand 20 September om 8.30 nm. in die Stadsaal, Durban, gehou word. Spesiale reëlings is getref om te verseker dat hierdie funksie die uitstaande hoogtepunt van die sosiale programme van die 1957-Mediese Kongres sal wees.

Bywoning sal tot 500 pare beperk wees.

BELANGRIKE KENNISGEWING

Weens die aantal aansoeke om kaartjies vir beide die Feesmaaltyd en die Dansparty, is toelating daartoe nou beperk. Kaartjies vir beide hierdie funksies sal in strenge volgorde uitgegee word soos die voornemekaartjies ontvang word.

Lede wat die Kongres gaan bywoon, word dus gevra om so gou moontlik aansoek te doen om teleurstelling te voorkom.

KONGRESFOOIE

Om ophoping te verhoed en om registrasie op die openingsoggend te bespoedig, word lede versoek om die registrasiefooi van £3 3s. (drie ghienies), die bedrag vir die Feesmaaltydkaartjies, £2 2s. (twee ghienies), wat alle wyn en sterk drank voor en gedurende die dinee sal insluit, en die bedrag vir die Danspartykaartjies, £3 3s. (drie ghienies) vir 'n dubbele kaartjie, indien moontlik voor die Kongresdatum, aan die Ere-Tesourier, Mediese Sentrum 53, Fieldstraat, Durban, te stuur.

B. Crowhurst Archer
S. Disler
Ere-Organiserende Sekretarisse,
41*ste Suid-Afrikaanse Mediese Kongres*

Mediese Sentrum 53
Fieldstraat
Durban

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES—TARIFF BOOK

Owing to unforeseen circumstances it has not been possible to issue the tariff book in time for the fees to become effective from 1 July 1957, as originally intended. A further notice regarding the publication of the tariff book will be issued in due course.

As the list of approved medical aid societies depends on which societies signify their acceptance of the revised tariff it can only be published at the time that the tariff book is published.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
26 June 1957

MEDIESE HULPVERENIGINGS—TARIEFBOEK

Weens onvoorsiene omstandighede was dit onmoontlik om die tariefboek betyds uit te gee om die gelde van 1 Julie 1957 af in werking te laat tree, soos oorspronklik bedoel was. 'n Verdere kennisgewing in verband met die publikasie van die tariefboek sal ter bekwamer tyd gegee word.

Daar die lys van goedgekeurde mediese hulpverenigings afhang van watter verenigings te kenne gee dat hulle die hersiene tarief aanneem, kan dit slegs gepubliseer word wanneer die tariefboek ook uitgegee word.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
26 Junie 1957

QUESTIONS ANSWERED

WOOD-DUST ALLERGY

Question

A wood-craftsman who depends on his craft for his livelihood, has become sensitized to imbuia and stinkwood sawdust. Not only does he develop an acute dermatitis on actual contact with the sawdust, but on inhalation it produces marked swelling of the face and eyelids, and all the symptoms and signs of an 'allergic hay-fever'.

Is it possible to desensitize him by a course of injections? Are any suitable antigens obtainable anywhere? Hydrocortisone ointment (2%) applied locally relieves the contact dermatitis, but I am afraid to use cortisone systemically for any length of time. What would you advise in this case?

Answer

A physician writes: Sensitivity to wood dusts is not a common form of allergy. Like 'baker's itch' and 'sugar boiler's dermatitis' it only seems to develop after a considerable exposure—usually a period of years rather than months. The reasons for this are not clear.

The ideal treatment is for the patient to change his occupation; this is patently impossible in all but a few cases; but it is often

well worth while to take the patient off his work until the skin condition is under control. The most effective immediate treatment is the local application of 2% hydrocortisone ointment. Thereafter there are two main lines of treatment.

1. *Protection.* The use of a barrier cream is well worth while. When exposure is at its worst, for instance when the patient is working at a lathe and is being sprayed with dust, it may be possible to persuade him to wear a mask. Spectacles with plain glass in them to ensure that the eyes are protected to quite an extent.

2. *Curative.* It is well worth while trying to desensitize the patient by means of extracts of the woods involved. Samples of sawdust should be sent to the South African Institute for Medical Research with a request for the preparation of suitable extracts for treatment. Personally I have found that a course of histamine desensitization by means of ionization has been very helpful. The amount of treatment given depends on each individual case; probably an average course would be 2 treatments a week for 6 weeks.

UNIVERSITY OF THE WITWATERSRAND: FACULTY OF MEDICINE EXAMINATION RESULTS

The following candidates have completed all the requirements of the Sixth Professional Examination for the degree of M.B., B.Ch.:

Baise, J.
Brondum, T. v. W.
Broude, A. M.
Chegwidden, M. J.
Donnolly, P. W. H.
Dreosti, A. V.
Giunchi, U.
Herbst, S. H. A.
Katz, R. T.
Lichtigfeld, F. J.
Makhene, E. R. W.
Mason, J. Y. J. M.
Mistry, B. D.
Morar, P.
Pillay, G. S.
Pswarayi, F. J.
Raftopulos, P.
Rajh, C. N. C.
Ramjee, A. K.

PASSING EVENTS: IN DIE VERBYGAAN

Leipzig Autumn Fair. It is announced that at the Leipzig Autumn Fair, which will be held on 1-8 September 1957, medical apparatus and laboratory equipment will be included amongst the goods displayed.

* * *

Dr. W. Silber, M.B., Ch.B. (Cape Town), F.R.C.S. (Edin.) has changed his address from National Mutual Buildings, Church Square, Cape Town, to 316 Medical Centre, Heerengracht, Cape Town. The telephones remain unchanged, viz. residence 7-4458, rooms 3-7785; if no reply 7-7727.

Dr. W. Silber, M.B., Ch.B. (Kaapstad), F.R.C.S. (Edin.) het sy adres verander vanaf National Mutual-Gebou, Kerkplein, Kaapstad, na Mediese Sentrum 316, Heerengracht, Kaapstad. Die telefone bly onveranderd, nl. woning 7-4458, spreekkamers 3-7785; indien geen antwoord nie, 7-7727.

* * *

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Mediese en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering.

Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Klerksdorp District Mines Benefit Society. The Southern Transvaal Branch Council announces that negotiations with this Society for the payment in full of all specialist fees having proved fruitless, the Branch Council has withdrawn the privilege of the Medical Aid Tariff for specialist fees with effect from 30 June 1957. From 1 July 1957 specialists may charge normal private fees.

* * *

Mr. Bernard Polonsky, M.Ch. Orth. (L'pool), F.R.C.S. (Edin.) has been elected a Fellow of the British Orthopaedic Association.

* * *

Mr. L. Blumberg, F.R.C.S. (Eng.) has moved to new consulting rooms at 1010 Medical Centre, Heerengracht, Cape Town. The telephone numbers remain, rooms 2-8800, residence 7-9548.

* * *

Dr. L. Blumberg, F.R.C.S. (Eng.) het die adres van sy spreekkamers verander na Mediese Sentrum 1010, Heerengracht, Kaapstad. Die telefoonnommers bly onveranderd, spreekkamers 2-8800, woning 7-9548.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van ernstige epidemiese siektes en poliomielitis in die Unie gedurende die tydperk 14 Junie-20 Junie 1957.

	Poliomiëlitis				
	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	—	4	—	—	4
Kaapprovinsie ..	1	4	7	—	12
Oranje-Vrystaat ..	—	—	—	—	—
Natal	—	2	1	1	4
Totaal ..	1	10	8	1	20

Pes, Pokkies, Tifuskoors: Geen.

DIFFERENTIAL SALARY SCALES

Resolutions on the application of differential salaries to non-European doctors in Provincial hospitals have been passed by the Natal Branches of the Association.

The Natal Inland Branch's resolution is as follows: 'The Natal Inland Branch of the Medical Association of South Africa views with the greatest concern the action of the Provincial Councils in discriminating against certain medical practitioners by paying them, not according to their skill, ability and experience, but according to their race. The Natal Inland Branch urges Federal Council to issue a statement of policy on the subject, which it hopes will include a strong protest against such a grave change of principle.'

The Natal Coastal Branch of the South African Medical Association at a meeting held on 25 June 1957 passed the following resolution: 'That this Branch disapproves of a salary scale discriminating against non-European doctors in Provincial hospitals and requests the Natal Augmented Executive of Federal Council to make representations on the subject to the Directors of the Provincial medical and health services.'

REVIEWS OF BOOKS : BOEKRESENSIES

BONE STRUCTURE AND METABOLISM

Ciba Foundation Symposium on Bone Structure and Metabolism. Editors for the Ciba Foundation. G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Cecilia M. O'Connor, B.Sc. Pp. xii + 299. 121 Illustrations. 45s. net. London: J. & A. Churchill Ltd. 1956.

Contents: Chairman's opening remarks. Structure of bone form the anatomical to the molecular level. Structure of bone salts. The histological remodelling of adult bone. An autoradiographic study. Fibrogenesis and the formation of matrix in developing bone. The mucopolysaccharides of bone. Autoradiographic studies of the formation of the organic matrix of cartilage, bone and the tissues of teeth. Uptake of ^{35}S in the differentiation and growth of cartilage and bone. *In vitro* uptake and exchange of bone citrate. The magnesium content of bone in hypomagnesaemic disorders of livestock. The mechanism of nutrition in bone and how it affects its structure, repair and fate on transplantation. Studies on the repair of fractures using ^{32}P. Metabolic studies on vitamin D. The mode of action of vitamin D. Variations in sensitivity to vitamin D: from vitamin D resistant rickets, vitamin D avitaminotic rickets and hypervitaminosis D to idiopathic hypercalcaemia. Present knowledge of parathyroid function, with especial emphasis upon its limitations. The indirect assessment of parathyroid function. Vascularity of bone in relation to pathological studies. Some observations on experimental bone disease. Osteodysmetamorphosis foetalis. Bone as a critical organ for the deposition of radio-active materials. General Discussion. Chairman's closing remarks.

To anyone who attempts to keep pace with the phenomenally widespread researches in bone growth and structure, with all its biochemical and biological ramifications and unique techniques, it is very obvious that much water has passed under the bridges since John Hunter recorded in 1778 that when the 'bony part' of the teeth was burned, it blackened and when its salts were removed by acid, there remained a 'gristly or fleshy' residue. New information is added so rapidly and constant revision of theories is so frequent that the nomenclature alone of a decade ago is today inadequate in many respects.

This book records the wisdom of the Ciba Foundation in once again bringing together leading scientists, on this occasion from 10 countries, to meet informally (in this case in cooperation with the Bone and Tooth Society of Great Britain) so as to present the trends of modern research, to discuss the difficulties, to suggest improvements in intricate techniques, and to come to some agreement on basic concepts.

The closing remarks of the Chairman of the Symposium pertinently state the case: '. . . we have covered a wide field of bone metabolism and growth, the structure, function and pathology, and we have seen what is going on in different aspects of this field. It seems to me that the anatomists and the histologists, the people, that is, who look at bone, are making faster progress than the rest of us. The biochemists are up against the problem of the sample, where to take it from, its purity and how to handle it. As for the clinicians, they are greatly handicapped by the unfortunate inability to obtain and study pure parathyroid hormone, and by their inability to assay it in biological fluids. As a result, they are making slow progress, as far as I am aware. I do not think we will get very much further until we settle more directly the question of which clinical situations really have got hyperparathyroidism, primary or secondary, and which have not.'

The reviewer discovered something new in each chapter, especially in the discussion at the end of each paper, and found the volume wholly inspiring and revealing. It must be regarded as an essential addition to the bookshelves of anatomists, biochemists (in particular), pathologists and clinicians actively interested in bone and bones.

The Ciba Foundation is to be heartily congratulated on another fine production.

R.S.

YEAR BOOK OF PAEDIATRICS

The Year Book of Pediatrics (1956-1957 Year Book Series). Edited By Sydney S. Gellis, M.D. Pp. 480. 134 Illustrations. $6·75. Chicago: Year Book Publishers Inc. 1956.

Contents: Introduction. The Premature and the Newborn. Nutrition and Metabolism. Infectious Disease and Immunity: Poliomyelitis. Tuberculosis. Allergy. Dentistry and Otolaryngology. Ophthalmology. Respiratory Tract. Gastrointestinal Tract. Genitourinary Tract. The Heart and Blood Vessels. Blood. Endocrinology. Orthopedics. Dermatology. Neurology and Psychiatry. Tumors. Therapeutics and Toxicology. Miscellaneous.

This Year Book, as in previous editions, contains extracts of many articles of paediatric interest, with occasional comments by contributors well known in their field.

An interesting feature in this edition, is the number of contributions made by South Africans. Dr. Robert McDonald's article on subcutaneous fat necrosis and sclerema neonatorum, is extensively quoted and favourably commented upon. There are summaries of two papers on myocarditis of the newborn and its association with Coxsackie Group-B virus infection: these are by Drs. Montgomery, Gear, Prinsloo, Kahn and Kirsch, and Drs. Javett, Heymann, Pepler, Lurie, Measroch and Kirsch respectively. Dr. H. Braude's case of phenylketonuria in a European child treated with a diet low in phenylalanine is also mentioned. In the orthopaedic section, Dr. A. Helfet's paper concerning a new way of treating flat feet in children, is discussed in detail.

These articles, except for the last, are fairly representative of the general selection, i.e., the accent is on paediatric problems encountered in hospital or in specialist practice. However, paediatrics occupies such a large part of the average medical practice that consideration should be given to advances in the management of the everyday problems that confront the doctor attending to sick children.

One would like to see the editors resume the system of providing short reviews to introduce the various sections, e.g. Nutrition, Infectious Diseases, Allergy etc. Such surveys, describing present activities and future trends, would be far more informative and stimulating than the rather dull comments presently in use.

I.M.

PAPERS ON THE CEREBELLUM AND ON VISION

Selected Papers of Sir Gordon Holmes. Compiled and Edited by F. M. R. Walshe. Pp. xii + 264. Illustrations. 20s. net. London: Macmillan & Co. Ltd. 1956.

Contents: Preface. Prologue. 1. Papers on the Cerebellum. 2. Papers on the Cortical Representation of Vision. 3. Papers on Visual Orientation and Attention. Bibliography of Published Works.

This small volume has been published by the Guarantors of the Journal *Brain* to honour Sir Gordon Holmes on the occasion of his eightieth birthday. On top of his remarkable career as a neurologist, Holmes was Editor of *Brain* from 1922 to 1937. A bibliography of his published works is appended at the end of the monograph.

Sir Francis Walshe in his introduction points out that many of these articles are no longer accessible but that the Guarantors of *Brain* found it impossible to reprint them all in one volume. Instead, 10 representative papers have been selected. These have been chosen from his major contributions to the pathology and symptomatology of cerebellar diseases, and from his physiological and anatomical studies on the cortical representation of vision, and on visual orientation and attention. Walshe comments further, 'Together they constitute an achievement in original observation and in the careful analysis of neural disorders in terms of normal function that has not been surpassed in our day, and they are now embodied in the corpus of established neurological knowledge.'

The best comment which can be made on the essays reprinted is to recall the mind and method of the author in terms of his own chosen quotation from Francis Bacon, 'For myself I found I was fitted for nothing so well as for the study of Truth; as having a mind nimble and versatile enough to catch the resemblance of things (which is the chief point), and at the same time steady enough to fix and distinguish their subtler differences; as being gifted by nature with desire to seek, patience to doubt, fondness to meditate, slowness to assert, readiness to reconsider, carefulness to dispose and set in order; and as being a man that neither affects what is new nor admires what is old, and that hates every kind of imposture.'

J.F.B.

MEDICAL LABORATORY TECHNOLOGY

An Introduction to Medical Laboratory Technology. By F. J. Baker, F.I.M.L.T., F.R.M.S., R. E. Silverton, A.I.M.L.T., F.R.M.S. and Eveline D. Luckcock, A.I.M.L.T. Pp. x + 330. 108 Figures. 34s. 6d. + 1s. 5d. delivery. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Limited, P.O. Box 792, Durban. 1955.

Contents: Foreword. Preface. Acknowledgements. *Section I. General.* 1. General Laboratory Glassware. 2. General Laboratory Apparatus. 3. Elementary Glass-Blowing. 4. Elementary Microscopy. *Section II. Chemistry.* 5. General Chemistry. 6. Elementary Colorimetry. 7. Urine. 8. Blood. 9. Examination of Cerebrospinal Fluid. *Section III. Histology.* 10. Fixation. 11. Decalcification. 12. Paraffin Wax Technique. 13. Section Cutting. 14. Biological Staining. 15. Staining Procedures. *Section IV. Bacteriology.* 16. Introduction to Bacteriology. 17. Sterilization. 18. Preparation and Use of Culture Media. 19. Anaerobic Methods. 20. Simple Agglutination Test. 21. Routine Bacteriological Examination of Specimens. 22. Care and Maintenance of Laboratory Animals. *Section V. Haematology.* 23. Introduction to Haematology. 24. Haematological Techniques. 25. Colour Index and Absolute Values. Appendix. Bibliography. Glossary. Index.

This book is meant primarily for technicians preparing for the Intermediate Examination of the Institute for Medical Laboratory Technology in Great Britain and covers the syllabus intended for that examination.

The book is very readable, the theoretical background is simply and clearly stated, and the techniques described are concise and well presented. There are innumerable useful suggestions and attention to important details is not overlooked.

Descriptions of laboratory equipment are well illustrated making it easy for the novice to grasp the essentials.

In the General Section centrifuges, syringes, pipettes, sterilizers, incubators, microscopes, balances and glassware are discussed fully, and there are several valuable tips on glass blowing.

There is a section in Chemistry in which compounds, mixtures, valency, gas laws, equivalents and pH are explained, visual colorimeters described and, of course, essential tests on urine and blood given.

A section on Histology discusses the various fixatives and preparation of tissues for section and staining procedures.

The section on Bacteriology is perhaps a little brief. Examination of stools for helminths is not discussed and serology is dismissed in one chapter.

There is a valuable chapter on the care, handling and inoculation of animals, and a section on Haematology which is a useful introduction to this subject.

In the Appendix are general notes and precautions as well as advice in the event of laboratory accidents.

This book, which is beautifully printed, should prove of value to the technician starting training.

G.S.

EMERGENCIES IN MEDICAL PRACTICE

Emergencies in Medical Practice. Fifth Edition. Edited by C. Allan Birch, M.D., F.R.C.P. Pp. xii + 684. 155 Illustrations 12 in colour. 37s. 6d. + 1s. 7d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1956.

Contents: I. The Emergency Bag. II. Acute Poisoning. III. The Hazards o Medical Procedures. IV. Acute (Non-Surgical) Abdominal Catastrophes. V. Other (Non-Surgical) Abdominal Emergencies. VI. Medical Emergencies in Obstetrics and Gynaecology. VII. Respiratory Emergencies. VIII. Cardio-Vascular Emergencies. IX. Emergencies in Blood Diseases. X. Fits, Faints and Unconsciousness. XI. Neurological Emergencies. XII. Psychiatric Emergencies. XIII. Medical Emergencies in Diabetes. XIV. Medical Emergencies in Other Endocrine Disorders. XV. Medical Emergencies in Renal disease. XVI. Medical Emergencies in Infancy and Childhood. XVII. Emergencies in Infectious Fevers. XVIII. Emergencies in Tropical Medicine. XIX. Emergencies in Industrial Medicine. XX. Medical Emergencies at Sea. XXI. Medical Emergencies in the Air. XXII. Ophthalmic Emergencies. XXIII. Emergencies in Skin Disease. XXIV. Emergencies during Anaesthesia. XXV. Bites and stings and Miscellaneous Emergencies. XXVII. Medico-Legal and other Non-Clinical Emergencies. XXVIII. Practical Procedures. Appendix I. Vaccines, Sera and Anti-Venines. Appendix II. Haemophilia. Appendix III. Special Centres for Poliomyelitis. Appendix IV. Night Blood Banks. Appendix V. Emergency Bed Service. Appendix VI. Corneal Grafting. Appendix VII. Drugs mentioned in the text and some of their equivalent preparations.

The fact that a new edition of this work has been brought out every 2 years since its first appearance in 1948 bears testimony to its popularity and usefulness. The emergencies dealt with cover every field both in general and specialist practice, and it is a work of reference which every practitioner (not excluding the surgeon) should carry in the cubby-hole of his car. Having read the volume from cover to cover I find much that is entirely off my beat, but very much on other people's.

The index is sufficiently large and well arranged to enable any particular subject to be found with ease.

The field covered is very extensive, and each part of it is dealt with by someone who is an expert in his subject. The opinions offered are personal opinions of methods tried out and found to be sound and valuable by the writers themselves, and the 25 contributors are all practitioners who are regarded as experts whose opinions are worthy of credence.

The value of this book is enhanced by the generous provision of illustrations, 155 in all, 12 of which are in colour.

This volume requires no recommendation to those who have already made its acquaintance; to those who have not, I would suggest that it should be regarded as a necessary part of their armamentarium.

F.R.L.

EAR, NOSE AND THROAT DISEASES

Symptome und Diagnose der Hals-Nasen-Ohren-Krankheiten. By Prof. Dr. M. Schwarz. Pp. VIII + 351. 88, Z.T. Mehrfarbigen Abbildungen. DM 48.-. Stuttgart: Georg Thieme Verlag. 1956.

Contents: Einleitung. Systematische Diagnostik. Symptome und Diagnose. Formveränderung der Ohrmuschel. Gestalt- und Reliefstörungen. Abstehende Ohrmuschel. Verlegungen des äusseren Gehörganges. Einengungen des äusseren Gehörganges. Das eingedrängte Trommelfell und die Funktionsstörungen der Ohrtrompete. Durchlöcherung des Trommelfells, ihre Begutachtung. Ohrenschmerzen. Ohrenlaufen. Der krankhafte Warzenfortsatz. Ohrensausen. Schwerhörigkeit. Schalleitungs- bzw. Mittelohrschwerhörigkeit. Schallwahrnehmungs- bzw. Innenohrschwerhörigkeit. Taubheit, einseitige, doppelseitige. Begutachtung. Schwindelerscheinungen. Gleichgewichtsstörungen und Spontandeviationen. Nystagmus. Begutachtung. Formveränderungen der äusseren Nase. Behinderte Nasenatmung. Nasenbluten. Schnupfen. Lidschwellungen und Verlagerungen des Augapfels. Schluckschmerzen. (orale, tonsillogene, pharyngeale, laryngeale, ösophageale). Schluckbehinderungen. Stimmstörungen. Begutachtung. Atemnot und Erstickungsanfall. Sprachstörungen. Kopfschmerzen. (rhinogene, dentogene, otogene, cervicale, meningeale; Liquordiagnostik). Der pyogene Allgemeinifekt. (otogen, rhinogen, tonsillogen) Focalinfekt. Lymphknotenschwellungen. Geruchstörung. Stauungspapille und Neuritis nervi optici. Abducenslähmung. Trigeminusstörungen. Facialislähming. Schulterlähmung. Zungenlähmung. Sachregister.

In the preface the author states that when faced by a sign or symptom one tends primarily to consider the commonest diseases. Eventually, however, all the rarer ones are considered too. Therefore it is important, not so much to summarize the typical features of ear, nose and throat diseases, but to study the signs and symptoms which arise in this branch of medicine with a view to coming to a correct diagnosis. With this in mind the author has made the subject of each chapter one of the main signs and symptoms of otorhinolaryngology. After describing in detail all the variations with which it may appear, he proceeds to furnish a full differential diagnosis. The manner in which the book is drawn up facilitates rapid gathering up of knowledge. It is a pleasure to consult it.

W.E.K.L.

ELECTROCARDIOGRAPHY

Clinical Unipolar Electrocardiography. Third Edition. By Bernard S. Lipman, A.B., M.D., F.A.C.P. and Edward Massie, A.B., M.D., F.A.C.P. Pp. 397. 325 Illustrations. 87·50. Chicago: The Year Book Publishers, Inc. 1956.

Contents: 1. Introduction. 2. Physiologic Principles. 3. Electrical Position of the Heart. 4. Ventricular Enlargement. 5. Bundle Branch Block. 6. Myocardial Infarction. 7. Abnormal Electrocardiographic Pattern. 8. Congenital Heart Disease. 9. General Comments on Interpretation. 10. Spatial Vector Interpretation. 11. Cardiac Arrhythmias. Illustrative Unipolar Electrocardiograms. Illustrative Electrocardiograms of the Cardiovascular Lesions Amenable to Surgery—Congenital and Acquired. Illustrative Electrocardiograms of the Arrhythmias. Ashman and Hull Tables. Bibliography. Index.

This book can be heartily recommended to all students and medical practitioners. It is not a comprehensive reference book and aims at nothing more than a description of the simple fundamentals of electrocardiography. To the uninitiated it should serve as one of the most useful books in this field of cardiology. In the initial chapters, a simple introduction is given to the fundamentals underlying electrocardiography and the physiological principles responsible for the curves obtained. The normal basic patterns associated with changes in position and the explanation of the five basic normal patterns of the heart are well demonstrated. The diagrams are simple and the text clear and concise.

A description of the basic abnormal patterns follows. The explanation of the derivation of these patterns is kept as simple as possible and no controversial detail is included. The graphs associated with ventricular hypertrophy, bundle-branch block and cardiac infarction can be readily understood without being complicated by unnecessary detail. An excellent account is given of vectorcardiography and so simply that the principles can be grasped in one reading. Being a small book, the arrhythmias are

encompassed in a few pages and only the common simple disturbances are discussed.

A third of the book is devoted to common patterns and, here again, in keeping with the text, only classical graphs are illustrated. The book should serve as a primer for all medical students and should cover all the usual problems of routine clinical electrocardiography.

V.S.

THE CONSULTING-ROOM ASSISTANT

The Office Assistant in Medical or Dental Practice. By Portia M. Frederick and Carol Towner. Pp. x + 351. Illustrations: 55 figs. $4.75. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: 1. A Career as an Office Assistant. 2. Medical Ethics and Medico-Legal Affairs. 3. The Right Kind of Reception. 4. Telephone Technique. 5. Appointments. 6. Handling the Mail. 7. Medical and Dental Records. 8. Filing the Records. 9. Medical and Dental Fees. 10. Extending Credit and Making Payment Arrangements. 11. Billing and Collecting Procedures. 12. Bookkeeping. 13. Banking. 14. Accident and Health Insurance. 15. Physical Examinations and Office Assisting Techniques. 16. Sterilization Techniques. 17. Care of Instruments. 18. Pharmacology and Preparation of Medications. 19. Administration of Medications; Injections. 20. Immunology. 21. Physical Therapy. 22. Housekeeping. 23. Supplies. 24. Library and Editorial Duties. 25. Professional and Health Organizations. 26. Daily Plan. Appendix 1. Suggested Readings for Reference Books for Medical and Dental Assistants. Appendix 2. Common Abbreviations and Symbols. Index.

All doctors will agree that a capable assistant is a great asset in running a practice.

There is so much useful information in 'The Office Assistant' that the average receptionist/assistant will, on reading it with care, acquire a training that, coupled with efficiency and experience, will make her well-nigh indispensable.

The average doctor would also profit by reading it and choosing methods which he feels would suit him and his type of practice. The fields covered are wide as the index given above indicates, and the human-relationships angle which can do so much to make or mar a practice in the outer office are given their important place.

Although the book was written primarily by Americans for Americans, there is much that can be learned from it that is common to all countries and types of practice.

A.H.T.

THE LABYRINTH

The Labyrinth. Physiology and Functional Tests. By Joseph J. Fischer, M.D. Pp. xi + 206. Figures 30. $6.00. London and New York: Grune & Stratton, Inc. 1956.

Contents: Foreword. Author's Preface. 1. General Physiology. 2. Applied Physiology. Spontaneous Manifestations. Induced Reactions. 3. Functional Tests. Spontaneous Manifestations. Induced Reactions. Evaluation of Abnormal Reactions. Differentiation of Peripheral and Central Lesions. References. Index.

Joseph J. Fischer writes with the authority one expects from an associate of Adam Politzer and Bárány, the latter a recipient of the Nobel Prize for his work in this field. The clinician, however, is bewildered by the excessive number of theories and the enormous difficulties in reconciling them. Every hypothesis put forward is disputed. Quite apart, therefore, from its difficulty of comprehension, the labyrinth is the cockpit of fierce controversy.

Fischer's book fills a great need in clearing the field of undergrowth. The book is cleverly arranged into sections of General Physiology, Applied Physiology, and Functional Tests, enabling the reader to find just what he requires without wading through endless pages of irrelevant material. Seldom has a book appeared that excels Fischer's work in simplicity of presentation and is so helpful to those wishing to refresh their knowledge or familiarize themselves, whether they be otologists or neurologists, with the highly complex functions of this organ. It is a superbly valuable aid in neurological diagnosis of brain tumours, intracranial complications, and differentiation between peripheral and central labyrinthine lesions or between a cortical and pontine location in disturbances of associated eye movements.

In the last decade, the labyrinth has moved into the forefront of research. The high speeds attained by jet-propelled aircraft, rapid acceleration and deceleration, high turning-speed, swift ascent and descent, exert a profound effect on the labyrinth.

Fischer's book is a splendid contribution to an understanding of these effects. The immense bibliography at the end of each section indicates the author's thoroughness in creating a work of this high standard.

O.O.P.

PAEDIATRICS

Pediatrics. Edited by Donald Paterson, M.D. and John Ferguson McCreary, M.D. Pp. xvi + 654. 192 Illustrations. £5 10s. net. London: Pitman Medical Publishing Co. Ltd. 1956.

Contents: 1. History Taking and Physical Examination. 2. Normal Growth and Development. 3. Diseases Due to Prenatal Factors. 4. The Newborn Baby. 5. Infant Feeding. 6. Fluid and Electrolyte Balance. 7. Nutritional Disturbances. 8. Diseases of the Alimentary Tract. 9. Accidents, Burns, Poisoning and Sudden Death. 10. Diseases of the Cardiovascular System. 11. Diseases of Ear, Nose and Throat. 12. Diseases of the Respiratory System. 13. Allergies. 14. Diseases of the Liver and the Pancreas. 15. Diseases of the Blood, the Spleen, the Lymph Glands and the Thymus. 16. Diseases of the Genito-urinary System. 17. Pediatric Gynecology. 18. Diseases of the Endocrine Glands. 19. Diseases of the Nervous System. 20. Mental Retardation and Behaviour Disorders. 21. Disturbances of Adolescence. 22. Diseases of the Skin. 23. Diseases of Muscles. 24. Diseases of the Bones and Joints. 25. Inborn Errors of Metabolism and Storage Diseases. 26. Diseases of the Eye. 27. Immunization. 28. Public Health Service. 29. Infectious Diseases. 30. Rickettsial Diseases and Those Caused by Helminthes, Arthropods and Protozoa. 31. Tuberculosis. 32. Syphilis. 33. Therapy and Procedures. Appendix. Index.

This is, from the geographical point of view, a new venture in paediatric textbooks. It is a composite work by a team of authors, largely of Canadian origin, under the leadership of the two named on the cover. Of these two one has for many years been known by name wherever reference to an authoritative text-book has been required and his association with this book infers that the contents will be of the highest quality. His partner in editorship is professor of paediatrics in the University of British Columbia.

There are, naturally, variations in the level of achievement in the different sections but there is very little in modern paediatrics that has not received at least honourable mention. Some more detail would have been acceptable in places, e.g. in the methods of resuscitating the newborn where the use of oxygen is recommended but no method of delivery suggested. The section on rheumatism is far from clear in its general format and the reader is left in doubt about what is meant by rheumatic fever, which term seems at times to cover everything rheumatic. The reviewer takes leave to doubt that the normal pulse rate of a year-old child is 100, which is elsewhere stated to be 120, and also that the emptying time of the stomach in the neonatal period is 6-8 hours. That kind of statement should surely have been questioned in a book of this calibre.

The book is intended for the daily use of the general practitioner and the preface indicates that it is meant as a compromise between the size of text-book for students and the large reference book. The claim is too modest. It is priced high but is good value. It can be unreservedly recommended.

F.J.F.

FIRST AID

J. F. Sutherland's First Aid to Injured and Sick. 46th Edition. Revised and rewritten by Halliday Sutherland, M.D. and I. Humphrey Evans, M.B., B.S. Pp. 85 + 46 Diagrams, 1 Coloured. 1s. 6d. net + 3d. postage abroad. Edinburgh and London: E. & S. Livingstone. 1957.

A book that has reached its 46th edition and of which 300,000 copies have been distributed since it first appeared in 1887 needs no pen to praise it. Its 85 pages, each only 2¾ by 4 inches in size, contain such a complete wealth of first-aid information as can only be realized on examination of the work itself.

It has been revised and rewritten by the original author's son, the well-known writer Dr. Halliday Sutherland, with Dr. Humphrey Evans as co-editor, and it is completely up to date in the methods and procedures described.

Its small size makes it possible to have it always conveniently at hand.

A.H.T.

'COMPEND' ADDENDUM FOR 1956

'The Compend' Addendum for the Year 1956. Compiled by W. Hetherington, F.P.S. Pp. 58. 4s. Bristol: John Wright & Sons Ltd. 1957.

New products are being added to the medical armamentarium with such frequency that this addendum to the 'Compend' for the year 1956 contains some 225 monographs.

There has been included, in addition, new information on many existing products which bring the original book up to date.

Those who appreciate the 'Compend' will want a copy of the 'Addendum for the year 1956'.

A.T.

PATHOLOGICAL HISTOLOGY

Pathological Histology. Fifth Edition. By Robertson F. Ogilvie, M.D., D.Sc., F.R.C.P.Ed., F.R.S.E. Foreword by A. Murray Drennan, M.D., F.R.C.P.Ed., F.R.S.E. Pp. xi + 482. 334 Photomicrographs in Colour. 52s. 6d. net + 1s. 5d. Postage Abroad. Edinburgh and London: E. & S. Livingstone, Ltd. 1957.

Contents: Foreword. I. Disturbances of Nutrition. II. Disturbances of the Circulation. III. Inflammation and Repair. IV. Specific Inflammations. V. Tumours. VI. Tumours (continued). VII. Circulatory System: Blood Vessels. VIII. Circulatory System: Heart. IX. Respiratory System. X. Alimentary System: Stomach and Intestines. XI. Alimentary System: Liver and Gall Bladder: Pancreas. XII. Urinary System. XIII. Reproductive System. XIV. Haemopoietic System. XV. Nervous System. XVI. Integumentary System. XVII. Locomotor System. XVIII. Endocrine System. Index.

Dr. Ogilvie's book is well known to undergraduate students in pathology and its popularity may be gauged by the appearance of 5 editions within the past 16 years. Its main value undoubtedly lies in its content of colour photomicrographs. With relatively few exceptions these are good and many of them are superb.

Detailed descriptions of the histological features are preceded by short accounts of the nature of the disease processes and of the macroscopic appearances of the diseased tissues. One has always felt that the text did not quite reach the high standard set by the illustrations and it is disappointing to find that certain deficiencies, and some errors, persist in the present edition. The author's classification of tumours (into simple, malignant and mixed) is hardly satisfactory. The mixed parotid tumour is an epithelial tumour and should not be classified along with nephroblastoma under the heading of blastocytoma. The views expressed on the mechanism of the chromaffin reaction are no longer acceptable. Otherwise lucid descriptions are sometimes spoilt by regrettable phrases such as 'polymorphs . . . which thither have been much facilitated in their emigration by the trelliswork of the superficial fibrinous exudate'.

In this 5th edition the number of illustrations has been increased by 39 and the text condensed by some 26 pages—a change in the right direction. Subjects discussed for the first time include currently popular conditions such as lupus erythematosus, scleroderma and temporal arteritis as well as old established entities like diphtheritic myocarditis, rheumatoid arthritis and osteitis deformans. Where old illustrations have been replaced by new, the change has usually been for the better, as e.g. the improved photomicrographs of bacillary dysentery (Fig. 194), tuberculous lymphadenitis (Fig. 67) and rheumatic endocarditis (Fig. 166). A separate index to the illustrations would be a welcome innovation in subsequent editions.

Despite one's criticism of the text, this book can be unhesitatingly recommended to the undergraduate student as a companion to his standard text-book of pathology. It will be of particular benefit if he studies it along with his slides. It should also prove of value to the postgraduate student revising pathology in preparation for a higher examination.

M.S.

ARMY MEDICAL SERVICES IN THE SECOND WORLD WAR

Medical History of the Second World War. Army Medical Services—Volume I—Campaigns. By F. A. E. Crew, F.R.S. Pp. xiii + 637. 91 Figures. XXXVI Plates. 92 Tables. 75s. net. London: Her Majesty's Stationery Office. 1956.

Contents: Prefatory Note by the Editor-in Chief. Book of Remembrance. Preface. 1: The Campaign in France and Belgium. 1939-1940. I. The First Phase. September 4, 1939-May 9, 1940. II. The Second Phase. May 10-June 2, 1904. III. The Saar and the Somme. IV. Review of the Work of the Army Medical Services. 2: The Campaign in Norway. April 15-June 8, 1940. 3: The Battle of Britain. July 10-October 31, 1940. 4: The Campaign in Libya. 1940-43. I. Initial Operations in the Western Desert. II. The Counter Offensive and the Pursuit to El Agheila. III. The Withdrawal to the Libya-Egyption Frontier. IV. The Health of the Troops. 5: The Compaign in Libya. 1940-1943. I. The Advance. II. The Siege of Tobruk. April 11-December 10, 1941. III. The Retreat. IV. The Health of the Troops. October 1, 1941-June 30, 1942. V. The Work of Certain Forward Medical Units. 6: The Campaign in East Africa. June 10, 1940-November 29, 1941. I. The Invasion of Abyssinia by way of Eritrea. II. The Invasion of Abyssinia by way of Italian Somaliland. III. The Health of the Troops. 7: The Campaign in Greece. April 6-28, 1941. 8: The Campaign in Crete. May 20-31, 1941. 9: The Campaign in Iraq. May 2-30, 1941. Chapter 10: The Campaign in Syria. June 8-July 14, 1941. I. The Invasion—June 8-July 14, 1941. II. The Occupation of Syria. 11: The Campaign in Persia. August 25-28, 1941. 12: The Campaign in Madagascar. May 5-November 6, 1942. 13: Malta. 1940-1943. Appendices.

This volume deals with the services rendered by the Army Medical Services in the campaign in France and Belgium in 1939-40 and in the ill-fated expedition to Norway. It goes on to deal with the medical methods used to cope with the indirect attack by Italy and Germany on the Commonwealth directed at severing the communications between Great Britain and the Dominions *via* the Mediterranean. This volume stops short at the time when Generals Alexander and Montgomery initiated the thrust which drove the Axis troops out of Africa.

The value of this historical record will be greatest to the student of military medicine. Since it is written in a factual style, making liberal use of accepted military abbreviations, reading is sometimes heavy-going. In spite of this, interest is well maintained. The strategical and tactical problems are clearly described, plans to deal with them are stated, and failures are recounted without excuses being offered. The lessons learnt are pointed out in critical summaries at the end of each chapter.

Praise is never invited and but sparingly given. If anyone doubts the ability of Army Administrative Medical personnel to rise to the needs of the sick and wounded soldier let him read these pages.

South African readers will find in detail the notable part played by the South African Medical Corps in East Africa, Abyssinia, Madagascar, Libya and on the Home Front. Those who participated in these campaigns may discover for the first time the parts which their units were playing in the various actions and, as a result, may be able to re-live their war years with added interest. In retrospect, too, they will regard, I am sure, the few criticisms made as being constructive and justified.

As a reference book this should be in every library.

R.L.F.

HIGH BLOOD PRESSURE

The Treatment of High Blood Pressure. Being the George Alexander Gibson Lecture for the year 1956 delivered in the Hall of the Royal College of Physicians of Edinburgh on 8th May by Frederick H. Smirk, M.D., F.R.C.P., F.R.A.C.P. Pp. 29. 3 Figures. 3s. 6d. Edinburgh: The Royal College of Physicians. 1956.

This booklet is a reprint of a lecture given in Edinburgh in 1956 and is little more than a brief introduction. It contains a review of the author's treatment of hypertension with the ganglion blocking agents for the most part. The indications are briefly stated and the method of treatment (and dosage schemes) with hexamethonium bromide, pentolinium and mecamylamine. The need for frequent blood-pressure readings is emphasized and stress is laid on an adequate dosage-regime. An inadequate dose is valueless and an excessive dose produces unpleasant symptoms from hypotension and parasympathetic paralysis.

V.S.

FLUOROSCOPY

Fundamentals of Clinical Fluoroscopy. With Essentials of Roentgen Interpretation. Second Revised Edition. By Charles B. Storch, M.D. Pp. xiii + 305. 318 Figures. $8·75. New York and London: Grune & Stratton, Inc. 1956.

Contents: Acknowledgements. Preface to the First Edition. Preface to the Second Edition. I. Basic Concepts: Mechanics, Protection and Dark Adaptation. II. Fluoroscopy of the Chest. III. Fluoroscopy of the Heart. IV. Fluoroscopy of the Pharynx, Hypopharynx and Esophagus. V. Fluoroscopy of the Stomach. VI. Fluoroscopy of the Small Bowel. VII. Fluoroscopy of the Colon. VIII. Intestinal Obstruction. IX. Fluoroscopy of the Gallbladder. Bibliography. Index.

This concise, clear and well-illustrated book, as its title implies, deals mainly with fluoroscopy. It does, however, stress that the fluoroscope does not replace the radiograph—this is often not sufficiently realized. Until electronic intensification is standard on all fluoroscopes—and expense alone places this well into the future—a radiograph should if possible be taken. Thus the clinician is armed with a clear and permanent record of the patient and not left merely to rely on the frailness of his visual memory.

The author also lays great emphasis on the need for adequate dark adaptation (not 'accommodation', as it is so often incorrectly called). He points out that average eyes going from the bright daylight to the fluoroscopic room require from 15 to 30 minutes of red goggles, or in the dark, before they acquire adequate rod vision. It would be interesting to know what percentage of non-radiological fluoroscopists first subject themselves to this inconvenience before 'committing' fluoroscopy.

The section on the oesophagus, especially in regard to the differentiation between a physiological phrenic ampulla and a pathological hiatus hernia, could be read with advantage by radiodiagnosticians as well as physicians and surgeons interested in minor variations from the usual in this region. All too often a phrenic ampulla is described as a hiatus hernia, and the label thus incorrectly attached proves a great liability to the patient.

In general there is little in this book that will be new to an experienced radiodiagnostician, but it should prove invaluable to the radiology student. One or two minor features might however surprise him. He would probably wonder how often the author, as he recommends, had found it necessary to use the vagina as a palpating base for the better fluoroscopic separation of loops of barium-filled large or small bowel.

Physicians and practitioners who do their own screening could not fail to benefit greatly from careful study of this work, and to them and radiology students it is strongly recommended.

W.J.L.

CORRESPONDENCE : BRIEWERUBRIEK

THE FREEDOM OF THE DOCTOR

About two years ago I wrote to you protesting against the gradual drifting of the medical profession into the hands of bureaucrats and Medical Aid authorities, and pleaded for strong action by Federal Council to prevent this.[1]

It is now obvious that matters are getting much worse and that we should act to get our freedom on a solid foundation. We ought to go so far as to arrange that even all full-time medical officers and teachers render accounts each month in lieu of salaries for services to whatever authority or institution employs them.

The only way in which we can save the dignity of the profession will be to keep it free and in our own hands.

Should there be a number of individuals in our ranks who are so deeply involved with the civil authorities that they do not wish to be free men any more, then they may form their own little association distinct from ours.

Further, it seems extremely desirable that all candidates for any office in our Association should before election publish in the *Journal* a resumé of their ideas about these very important matters.

As things are going now we all may soon be at the mercy of every farmer or country attorney who is appointed to a Government post.

G. P. Fourie

S.A. Mutual Buildings
Parliament Street
Cape Town
25 June 1957

1. Fourie, G. P. (1955): S. Afr. Med. J., **29,** 268.

RE: EDITORIAL ON IONIZING RADIATION

To the Editor: We consider the recent editorial[1] on 'ionizing radiation' to be a poor one, and one not calculated to inform the South African medical profession about recent advances in knowledge on the subject of the hazards of diagnostic radiology and their control. It seems proper to us, as radiologists, that an editorial in our Medical *Journal* should attempt to guide the profession in a rational attitude to diagnostic radiology. This we feel, the editorial signally fails to do.

The editorial contains several misstatements of fact, and also confuses legitimate risks of therapeutic radiation with risks which it is desirable to avoid in diagnostic radiology. Examples of the former errors are the statements that 'public health regulations of the most stringent kind are in force,' and 'the soft tissues of the foot do not show up under the fluoroscope'. There are several others. An example of the latter confusion is the statement, 'apart from the immediate local effects in the area irradiated, profound effects are registered on the whole body, and they are most profound on the reproductive cells'. There are no known local effects in the area irradiated at diagnostic levels of radiation.

Your editorial contains three suggestions aimed at reducing radiation risk:

1. That we should stop and think before subjecting a patient to radiation. In private practice at any rate, a referring doctor will certainly stop and think before sending a patient for an examination which, in the nature of things, involves financial outlay. Better use of essential X-rays would be achieved by more consultation with the radiologist. He is best qualified to advise what nature of examination can solve the clinical problem and, equally important, what the limitations of radiology are in a particular problem. The radiologist is usually in the best position to judge how to conduct an examination once he is armed with the clinical data. With this approach planned examinations directed towards elucidating specific problems, using as little radiation as possible, become feasible.

2. That legislative measures be taken to eliminate machines which are faulty, or which are so constructed that filters can be readily removed. This statement is probably made in confusion with therapy X-ray machines where such filters, if omitted, may result in serious tissue damage. Filters, while of value in reducing dosage in diagnostic radiology, could hardly receive a high priority as a subject of radiation legislation. Legislation against faults that machines may develop is hard to conceive. Perhaps certain standards could be insisted on before machines are imported. However, the subject of radiation legislation is a difficult one, and the desirability in South Africa of a legislative approach, as opposed to an advisory approach, is by no means established.

3. Screen and film sensitivity increases. While these factors may well be improved in the future, no immediate reduction in total population radiation level is to be anticipated from them.

There are certain factors which could reduce radiation levels in the immediate future and others which promise only a delayed reduction. Perhaps the most effective immediate means of reducing dosage would be more effective consultation between the radiologist and referring doctors, and planning of examinations by radiologists to give maximum information for minimum amounts of radiation.

Factors which will reduce radiation more gradually are the following:

A. Better training of radiographers. Two subjects need to be taught with greater emphasis than at present. These are surface anatomy, so as to permit much more accurate collimation of the X-ray beam when visualizing a particular anatomical structure (this is today regarded as the single most effective measure in reducing radiation), and radiation hygiene as it concerns both patients and operators.

B. Training of those who have to use fluoroscopes in the principles of safe fluoroscopy.

C. The image intensifier. Useful, in fact revolutionary, developments may be anticipated under this heading.

Better results may be anticipated from improved handling of rays rather than by technical improvements or legislation.

M. H. Fainsinger
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25 June 1957

1. Editorial (1957): S. Afr. Med. J., **31,** 580.

INTRAPROFESSIONAL ETHICS

To the Editor: I note with interest your sub-editorial on interprofessional ethics but I feel very strongly that *intra*professional ethics could do with a little spring cleaning.

As Medical Officer to a boys' preparatory school I experience

almost daily instances of lack of courtesy from my professional colleagues. This unfortunate state of affairs is most noticeable at the beginning of a new term, when numerous boys arrive with bottles of medicine, boxes of tablets or capsules, and even injections, with a verbal request that their treatment be continued. Never is there a letter from the family doctor. I say never, but there was this one letter: 'Dear Doctor, I have given Brown a bottle of mixture please see that he gets a dessertspoonful three times a day. Yours faithfully, X.Y.Z., M.B., Ch.B.'

After all I have the responsibility for the care of these boys for the next ten weeks or so and I should like to know what I am supposed to be treating and what the various tablets etc. are. It is manifestly impossible for me to interview every parent. In any case, many of the boys are unaccompanied. A note from the doctor would be very helpful.

It works the other way, too. We have the pernicious custom of 'free week-ends', when the boys are allowed out in the custody of parents or relations. Somebody thinks Johnny is looking a bit pale and he is rushed in to the family doctor, who suggests that he stays at home for a day or two and then sends him back complete with a course of treatment—again without any letter. Surely if Johnny is suffering from some non-obvious malady it would be better for the school authorities and myself to be made aware of the fact so that we can be on the look-out for possible developments.

I recently diagnosed a case of erythema nodosum and suggested that the child should be seen by a specialist. My advice was ignored and he was taken home to his own doctor who sent him back with a bottle of mixture and a message to the effect that the condition was not serious and would clear up in a day or two. It didn't; and the child was whipped away again and has not so far returned to school, but I have heard indirectly that the diagnosis has been confirmed.

A few weeks ago I suffered another insult. I use the word advisedly, because so frequently the implication is there that one is only a country doctor—and what else can be expected? I had a child under observation who had a lesion on his foot which I was becoming convinced came under the heading of 'dermatitis artifacta'. Again I advised specialist consultation, but the child was taken home and put into the hands of a G.P. whose first remark as conveyed gleefully to me was, 'A gross case of overtreatment'. The child was put into a nursing home and there he remains. His previous treatment had consisted of occlusive dressings, which cleared the condition immediately, alternating with no treatment at all, when the condition recurred within 48 hours! A telephone call to me by the other doctor—I don't know him—would have put him in the picture immediately and avoided considerable trouble for all concerned, especially the boy.

Yes, Sir, I think some intraprofessional spring cleaning would be very much in order.

Rural G.P.

21 June 1957

ARTERIAL ANASTOMOSIS AND ANGINA PECTORIS

To the Editor: Having had cause recently to revise the subject of arterial collaterals, I should appreciate the opportunity to communicate to your readers the results of my random thoughts on this subject.

Anastomosis between arteries may be of two main orders: (1) Junction of major branches of arteries, e.g. in the gastro-intestinal tract, and (2) between arterioles of magnitude 50-200 μ. The former type are uncommon but the latter universal (except in end-arteries) and it is on the integrity of these un-named smaller vessels that the collateral circulation to parts deprived of their normal blood supply depends.

When an artery is suddenly occluded, the regions distal to the point of obstruction usually evince signs of ischaemia in spite of the liberal distribution of connecting arterioles. I consider unnecessary the glib and parrot-like invocation of 'spasm' of these vessels as an explanation of their apparent non-functioning. Such a mechanism would be totally unphysiological, contrary to the laws of survival, and at variance with experience in other related fields.

There is admittedly a lag period of a few hours before the vessels dilate to allow maximum passage of blood. At the time of the vascular insult the walls of these arterioles, in common with those of all other muscular vessels, are in a state of normal tonus resulting from sympathetic stimulation and therefore cannot be prepared to cope with the extra volume. Their eventual dilatation is probably brought about by humoral means—accumulation of metabolites and, possibly, the stimulus of anoxia. I find it difficult to conceive of a bodily response which would result in closing of channels by which toxic substances could be removed.

My second point is concerned with the collateral circulation in angina of effort. As the arterial anastomoses are of the second order mentioned above, they are ineffective in limiting the area of infarction in acute coronary-artery occlusion. In angina, however, the obstruction is of a chronic nature and is not complete. Moreover, the patient is provided by the disease process with an excellent warning device which informs him, most effectively, when he is exceeding his bounds. The metabolites which probably cause the pain simultaneously provide the stimulus for increase in the diameter of the anastomosing arterioles. Two conclusions stem from this:

1. The longer a patient has had angina, the less extensive is an infarction likely to be when it supervenes.
2. If an angina sufferer manages to evade a complete coronary occlusion, he may, in the fullness of time, be spontaneously cured, because his arterial system has compensated for the obstruction, that is to say his attacks of pain will occur with diminishing frequency and his exercise tolerance will improve.

The practical corollary of this is that restriction of an angina patient's activities is a worthwhile prophylactic measure and he should be taught to have respect for the red light.

I. G. Schraibman

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19 June 1957

A STATE-AIDED INSURANCE SCHEME

To the Editor: There can be little doubt that in the interest both of the public and the profession alike, free choice of doctor with payment for services rendered on the lines of approved Medical Aid Societies, is the ideal scheme for medical practice.

On a conservative estimate, however, up to 50% of the European population in this country cannot afford this system of practice. It must, therefore, remain an ideal until such time as by legislation the State or employers of labour, or both, are compelled to contribute 50% of the premiums required for such an insurance scheme to work for all income groups. It is an established fact that Medical Aid Societies subsidized on a £ for £ basis function most effectively.

It would appear that the Association must continue to co-operate with Benefit Societies, who cater for lower income groups, whilst it is left to the individual or groups of individuals to provide their own arrangements for medical services.

It is generally agreed amongst those who have had the most experience of working for Benefit Societies, that a completely open panel for all medical services, including all the different specialities, is unworkable and quite impracticable.

The insistence of the Medical Association on open panels and free choice of doctor in all Benefit Societies might result in the grave disruption or even discontinuance of the Mines Benefit Society and Railway Sick Fund, with the possibility that the State might be forced to intervene and introduce medical services on the lines of the National Health Service in England, where it has been stated that the average general practitioner with a panel of 2,200 individuals on his list receives 2s. 6d. for each consultation.

Is not the time ripe for the Medical Association to start a campaign to educate the public to appreciate the benefits of a State-aided compulsory insurance scheme, on the lines of the Norwegian medical services? (*vide British Medical Journal*, 11 May 1957) and to convene a meeting between representatives of the Medical Association, the Government, Industry, Mines and Commerce to discuss the development of health services on these lines.

C. A. H. Green

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20 June 1957